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OSTEITIS FIBROSA AND OSTEITIS DEFORMANS.[1]

By H. R. SEAR, M.B. (Sydney),
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IN this paper it will be my endeavour to discuss briefly the types of osteitis met with under the terms *osteitis fibrosa* and *osteitis deformans* and also to deal shortly with the differential diagnosis of these lesions.

I hold that these two conditions merge into one another, a view held by many previous writers, one of whom coined the term *osteitis fibrosa deformans* to include all the types of these lesions.

The discussion will be considered more fully later.

One of the greatest difficulties that besets a radiographer is the absence of confirmation of many of his diagnoses. A large number of cases diagnosed by him as *osteitis fibrosa* and still more of those diagnosed as *osteitis deformans* do not warrant surgical interference and therefore the ultimate diagnosis, that of the histologist, is wanting.

My attention was first drawn in 1912 to this lesion, *osteitis fibrosa*, by Bythell and Barclay.

Their cases would now probably be classed as *osteitis deformans*. They mention that these were mistaken for syphilitic lesions. This is a mistake that may occur especially in the monosteitic type of lesion, but it is not likely to occur in the generalized type.

Many other people have since reported cases showing apparently the two lesions, *osteitis fibrosa* and *osteitis deformans* in the same patient, notably the first case reported by Hartung in 1914, also Southgate, Hartman and Klestadt.

Hansen reports a case which he thought showed a transition form. This I feel I must regard more as being in the *osteitis fibrosa* group.

Some authorities, such as Kolisco, are prepared to group the two lesions together; others, for example, Latzko, hold that they are two distinct and separate lesions. Holzknecht even holds that they should never be mistaken for each other. Latzko states that, however similar the two conditions are, yet they are distinct processes.

Hartman suggests that they are not basically different, but different expressions of the same pathological process; more of this later.

In this country cases usually styled *osteitis deformans* are quite common. We have seen as many as three patients with this condition in one day at Royal Prince Alfred Hospital. I have seen as many as five in a fortnight in my rooms. All told, I should think I have seen well over two hundred patients suffering from *osteitis deformans*. This seems an enormous figure, when one still reads the reports of individual cases in journals. It would appear that this lesion is much commoner in Australia than in most other countries.

Likewise in Australia bone cysts are not extremely uncommon; but the generalized type, more commonly termed *osteitis fibrosa* or *osteitis fibrosa generalisata*, is much rarer.

As I read through the literature at my disposal I came across the statement written in 1916 that since Paget wrote in 1877 two hundred and seventy odd papers have been written on that subject and that the letterpress of the one would do for that of any other; and so also the illustrations of one for the other. I began to wonder if I was only adding to the total of therefore unnecessary articles and if my views were actually worth recording. But in view of the fact that this paper deals with a very large number of cases I thought it worth while persisting in my endeavour.

My main conclusions are put at the end.

It is neither my province nor my intention to discuss in any detail the pathology of this lesion, but I cannot avoid saying a few words on the process with which I am dealing.

Knaggs describes *osteitis fibrosa* as a disease where the osseous framework and marrow are replaced by fibrous tissue. This description would also serve for *osteitis deformans*. The essential features histologically are the same for the two diseases:

(i) The disappearance of the original bone.

(ii) The substitution of a vascular connective tissue in place of it and of its intertrabecular marrow.

(iii) The formation of new bone from this connective tissue.

He states that *osteitis fibrosa* differs from *osteitis deformans* in that: (i) there is in *osteitis fibrosa* a complete absence of fat cells, (ii) there is a more uniform construction of the metaplastic bone in *osteitis fibrosa*, (iii) cysts and cystic formations are commoner in *osteitis fibrosa*, even if they occur in *osteitis deformans*.

Knaggs states that the two conditions most closely approach each other in the skull bones; radiographically this would appear to be the site where they show most difference. The skull changes in *osteitis deformans* may vary greatly, but I have never seen the woolly osteosclerosis of the cranial bones typical of *osteitis deformans* in a case of the *osteitis fibrosa* group.

Lotsch states that this proliferating and destructive fibrosis of the bone marrow, replacing the bone marrow, may lead to the formation of large fibrous masses (hence cases of fibromata), that by liquefaction we may get the cystic areas and that these masses may assume the appearance of giant-celled tumours. The differential features histologically appear to be a matter of degree rather than different processes.

On looking through the various textbooks on radiology to which we radiographers had to turn for guidance at the start, one must be struck with the paucity of help on this subject. For example, take the small book written by Bythell and Barclay

[1] Read at a meeting of the sections of Pathology and Bacteriology of Surgery and of Radiology at the Second Session of the Australasian Medical Congress (British Medical Association), Dunedin, on February 9, 1927.

in 1912 and compare it with the textbook on bone diseases by Baetyer and Waters written in 1923; in that time practically nothing has been added.

The usual classification in textbooks on radiology is into three groups: (i) Cysts, (ii) *osteitis fibrosa*, (iii) *osteitis deformans*.

Turning now to Bloodgood's classification of *osteitis fibrosa;* cysts are grouped under the heading *osteitis fibrosa*, the classification being as follows:

1. Single cysts with no connective tissue lining (in these we have this new connective tissue extending between the bone lamellæ of the surrounding shell of of bone);

2. Cysts with connective tissue lining, which lining is identical in structure with the connective tissue in walls of shell of group 1;

3. Small cyst or cysts in solid mass of fibrous tissue;

4. Solid mass of fibrous tissue (including fibroma);

5. Multilocular cysts.

Clinically Knaggs would divide *osteitis fibrosa* into four groups: (i) Those in which the lesion is represented by a solid mass of fibrous tissue, (ii) those in which the solid mass is showing signs of degenerating into one or more cysts, (iii) cases in which much bone is developed and the disease shows signs of being arrested, (iv) single cysts.

Radiographically I should like to classify the subgroups of *osteitis fibrosa* as:

1. Solitary cysts, with or without trabeculation;

2. Multiple cysts, such as Hansen's case and Isbister's rib case;

3. A somewhat cystic condition, sometimes involving one bone, sometimes many, approaching on the one hand the cyst, either single or multiple, and on the other passing through varying degrees of osteosclerosis till it approaches more closely the type of lesion seen in *osteitis deformans;*

4. A fourth group, characterized by a finely stippled, pitted or striated appearance, found most commonly in the jaws, where it has been classified by Victor Horsley as *leontiasis ossea;* this form has been included by Knaggs in one of the subgroups of the osteitic type of leontiasis.

This type of lesion, though found most commonly in the jaw, either superior or inferior, may occur, though rarely, in other bones, for example the tibia; here the appearance may approach very closely the type of lesion described in this country first by Cleland and later by Breinl and Priestly as boomerang leg.

I maintain that at one end of the series we have the typical cases of *osteitis fibrosa cystica generalisata*, as in Hansen's and Hartung's cases; that this group passes gradually through the semi-stringy, semi-cystic type, as in Dr. Royle's case of humerus and clavicle, in which the skull appeared normal, through Barclay and Bythell's case of 1912 to the class of cases usually styled *osteitis deformans*. Barclay's case would probably be styled *osteitis deformans* by many authorities at the present time.

On the other hand we have the class of *osteitis fibrosa generalisata*, such as described by Harris, which shows changes at the ends of the diaphyses that might almost form a transition stage to the condition known as "marble" bones. This type also helps to link up *osteitis fibrosa* with *osteitis deformans*.

Again compare the changes in *osteitis fibrosa* of jaws (one of the subgroups of the *osteitis* type of *leontiasis*) with the type of tibia shown in boomerang leg where the even stippling is much more suggestive of *osteitis fibrosa* of jaws than of the irregular striation seen in *osteitis deformans*. This forms the other end of the series of changes seen in these lesions.

It would appear that cyst formation in bone is never primary: it may arise by: (i) Liquefaction of fibrous tissue, hence *osteitis fibrosa cystica*, (ii) liquefaction of cartilage, which would possibly explain cases like Coles's case of fibro-cystic tumour of the patella, (iii) absorption of bone, possibly necrotic, in cases of osteo-arthritis, as described by Arbuthnot Lane; this class does not enter into this discussion.

Radiographically we are handicapped by the fact that cartilage, fibrous tissue *et cetera* are each as transradiant as cystic areas; in dense areas of osteosclerosis in *osteitis deformans* we may see transradiant areas, simulating cysts, but we cannot say whether these areas are occupied by fluid, fibrous tissue or cartilage. Operations or autopsy alone can determine this question and some pathologists would doubt the occurrence of cysts in *osteitis deformans*. This, however, is a question that can rarely be verified, as in the majority of the patients with these areas in *osteitis deformans* interference is not warranted.

Knaggs holds that the term *osteitis fibrosa cystica* should be reserved for those cystic conditions arising from degeneration of the fibrous tissue which has replaced a part of the original bony structure.

Turning now to the differential diagnosis, it would seem to depend finally upon the radiographer and the pathologist. In practice the former has to decide in quite a large proportion of the cases.

Upon what, then, are we to depend for our differential diagnosis? I think that the differential diagnosis is best attempted by considering the subgroups I have suggested individually, but first I should like to say a little about the differentiation between *osteitis deformans* and *osteitis fibrosa*.

This question is one that has perplexed radiographers for many years. Even as late as 1923 Knaggs in his Hunterian lecture says that the best differentiation for the skiagraphist is the age of the patient; if occurring in a young patient it is probably *osteitis fibrosa* and if in an elderly patient *osteitis deformans*.

For the radiographer to base his diagnosis in this way upon the age rather than the actual characteristics in the skiagrams leads nowhere and in fact

is highly unsatisfactory and inaccurate; for among von Recklinghausen's cases are, I believe, two females, one of forty-three and another of sixty-six. So also Hartung's first case of apparently mixed *osteitis fibrosa* and *osteitis deformans* was in a female of thirty-four and his third case of multiple cysts was in a male of thirty-seven; also Hansen's first patient was a female of forty-nine. Again, though *osteitis deformans* is a disease commonest in middle age, yet one case has been reported in a boy of sixteen and several have been reported in persons as young as twenty-one; the earliest case which I thought showed evidence of changes typical of *osteitis deformans* was in a boy of seventeen. Taking now the individual subgroups, I shall endeavour to see from what other lesions they have to be separated and if it is possible to do so.

Once again I should like to say how impossible it is in many cases to say what type of material is occupying a transradiant area in the bony tissue; fibrous, cartilaginous, myxomatous tissue, all are equally transradiant and cast no more shadow than cystic areas.

Group I.—Single Cysts.

Single cysts may be confused with fibroma (Hickey, 1916), myxoma, sarcoma, chondroma, myeloma, residual cartilaginous areas, solitary secondary deposits, as in hypernephroma, parasitic cysts and in rare cases bone abscess (especially tuberculous). Radiographically it is often impossible to separate these lesions; sometimes the site and possibly the age may be of some help. In children under eighteen Bloodgood says that a single lesion in the shaft usually means bone cyst, that multiple myelomata may develope at this age and that central giant-celled tumours are rare at this age.

In adults lesions of the shaft are usually chondroma, myxoma, myeloma, myxo-chondro-sarcoma or metastatic tumour. Bloodgood holds that giant-celled tumours in the shaft of adults are rare.

Central lesions in the epiphysis, rare in children, are usually giant-celled tumours; cartilaginous residues are, I think, more common than is usually supposed. In adults such solitary lesions in epiphyses are usually giant-celled tumours, with a few cases of bone cyst; they may also be chondroma, myxoma, myxo-chondro-sarcoma, metastatic tumours and multiple myeloma and in rare cases fibroma which should be grouped with cyst as *osteitis fibrosa*.

Group II.—Multiple Cysts.

Multiple lesions in the shaft are in children usually multiple bone cysts; in adults they are possibly multiple bone cysts, possibly multiple myelomata, sometimes multiple secondary growths. The typical case of multiple enchondromata is usually easy to differentiate; but sometimes we have cases probably of the nature of enchondromata which may closely approach the appearance of *osteitis fibrosa* (as in a patient seen at the Royal Alexandra Hospital for Children).

Group III.

In the third group the main differentiation is from *osteitis deformans* and from osteo-malacia. The most typical type of lesion in *osteitis deformans* is that usually seen in the pelvis and spine and here it is wonderfully constant in type. The vertebræ and the pelvis are very common sites of this lesion. I see many patients sent for radiographic

LEGENDS TO ILLUSTRATIONS OF THE ARTICLE BY DR. H. R. SEAR.

FIGURE I.
Paget's disease of the pelvis, showing marked bony overgrowth and thickening and the typical loose trabeculation. Note the decreased, unusually triangular true pelvis and the thickened overhanging superior part of the acetabular rims.

FIGURE II.
Osteitis deformans of the tibia showing loose and very irregular trabeculation and areas of increased trans-radiancy. Note especially the large transradiant area along the anterior border of the tibia towards the lower part of the lesion, probably representing a large mass of fibrous tissue.

FIGURE III.
Osteitis deformans of the fibula, a bone rarely involved. In this patient the fibula and *os calcis* were the only bones involved. The trabeculation here is much more even and "stringy," the fibula is very greatly expanded, almost equalling the tibia in size.

FIGURE IV.
Osteitis deformans of the tibia, the trabeculation here being much more uniform and even and the bone more compact.

FIGURE V.
Osteitis of the tibia of a most unusual character. The structure is extremely uniform and fine and the radiograph is practically identical with that of the mandible in Figure XI, which is a case of *osteitis fibrosa*; this tibia has been considered to be one of "boomerang leg," though the patient was not an aboriginal.

FIGURES VI and VII.
The clavicle and ulna of Dr. Royle's patient. Here the lesion is more cystic, though the condition must be classed as *osteitis deformans*. The skull in this patient showed no change.

FIGURE VIII.
Osteitis, sharply limited to the lower end of the shaft of the humerus, with some almost cystic areas, illustrating the type of case that opens up the discussion as to whether the lesion started as *osteitis deformans* or whether it is the result of an old cyst or fibro-cystic area of childhood.

FIGURE IX.
Osteitis deformans limited to the anterior edge of the tibia, sharply defined, with some very transradiant areas. This lesion has been observed for some six years and has shown practically no advance in that time.

FIGURE X.
Osteitis deformans of the lower edge of the mandible; apparently an unusual bone to be attacked by Paget's disease, for only two cases have occurred in this series. This bone belongs to the New Guinea skull described by Professor Burkitt in the *Journal of Anatomy*. Compare the structure here with that of the mandible in Figures XI and XII.

FIGURE XI.
The mandible of a male, aged 46; note the enormous depth of this mandible, especially in view of the absence of teeth. Compare its structure with that of the tibia in Figure V.

examination of the urinary tract who have the early changes typical of this condition, although it is not suspected.

This group may vary greatly; time was when I would boldly classify this as *osteitis fibrosa* and that as *osteitis deformans*, but as time has gone on and more and more cases have come under observation, some inclining towards the cystic group (Dr. Royle's clavicle and ulna case) and others towards the lesion styled *osteitis deformans*, I have reached a point at which I cannot differentiate the conditions and I hold that these two lesions merge into one another. As mentioned before, the two lesions have on several occasions been shown to occur in the same patient. From osteo-malacia the differentiation may be difficult and again in some cases of osteopsathyrosis the changes may approach the appearance sometimes seen in *osteitis fibrosa;* here the age, the general lack of calcium salts with the consequent transradiancy and the repeated fractures may be a guide.

Group IV.

The solid evenly stippled type, commonest in jaws, is regarded by some authorities as forming one of the subgroups of *leontiasis ossea*, for example by Knaggs and Bochenheimer; though this view is opposed by others, notably by Frangenheim. This has to be separated from specific lesions, but its structure is much more uniform and is usually very typical. In the tibia this type approaches the condition known as boomerang leg and through it *osteitis deformans.*

On reviewing all these cases I cannot help feeling, as others have done, that under the two headings *osteitis deformans* and *osteitis fibrosa* we are dealing with one pathological entity. My main reasons are in brief:

1. Histologically the difference appears to be one of degree, that they may really represent two stages of the one lesion.

2. Radiographically they appear to merge one into the other.

3. Clinically, though one is usually seen in youth and the other in middle or old age, we have no knowledge of the end result of the one or the start of the other. Are the fibro-cystic changes seen in youth to leave no changes behind or may they not later leave changes through hæmorrhage and organization that might be thought later to be *osteitis deformans?*

4. The two conditions may occur in the same patient.

5. There is a type of case, usually seen in the upper end of the humerus or of the tibia or of the femur, observed in later life, in which the lesions are sharply limited and therefore differ in that respect from the usual form of *osteitis deformans.* This form is usually classified as *osteitis deformans*, but it seems doubtful whether it could not be explained as being the end results of fibro-cystic disease in children, for these are common sites of cystic changes in children.

This, of course, is mere conjecture. Should these cases be called "late" *osteitis fibrosa* or merely *osteitis deformans?* Possibly time will show. I have had the opportunity to observe some of the conditions for several years; they would appear to be extremely slow in advancing and at the rate of their growth it is probable that they started when the patient was quite young, though the exact nature of the start of the lesion is unknown.

LEGENDS TO ILLUSTRATIONS OF THE ARTICLE BY Dr. H. R. SEAR.

Figure XII.

The mandible of a girl of approximately 17 years. A large part of the inferior border of the mandible was removed surgically, owing to an erroneous diagnosis of sarcoma. Note the fine stippling of the bone and the interference with the development of the teeth, not infrequent in this condition.

Figure XIII.

A localized area of *osteitis fibrosa* (somewhat magnified, to demonstrate the fine even stippling). This is the same type of structure as seen in the previous mandible, but better displayed by the enlarging.

Figure XIV.

Osteitis fibrosa cystica, with fracture.

Figure XV.

Cystic disease of middle phalanx. In such a case it is impossible radiographically to diagnose between this condition, hydatid and myeloid sarcoma.

Figure XVI.

Transradiant area at base of middle phalanx; in this case the pathological report was myxo-sarcoma.

Figure XVII.

Fibro-cystic disease of the upper end of the tibia; there has been no advance of the lesion in two years.

Figure XVIII.

Osteitis fibrosa cystica generalisata, referred by Dr. Isbister in a woman of young middle age. When first examined by me some eight years before these skiagrams were taken, the lesion was confined mainly to the lower ribs; now these ribs have mainly collapsed and the ribs above are the most expanded.

Figure XIX.

Skiagram of one of the humeri of the same patient; the lesion now is characterized by somewhat more transradiant areas extending right across the shaft. The condition is more evident than was the case at the time of the previous examination. This type of lesion appears to be very rare.

Figure XX.

Osteopsathyrosis; unfortunately only the femora, tibia and fibula of this patient were examined by X rays; numerous old fractures were present in all these bones. Note the loose trabeculation and uneven expansion, which may be present in this condition, though it is not essential. Note also how, when the trabeculation and expansion are present, the radiographic appearance tends to simulate *osteitis fibrosa cystica.* This type of structure was not present in the tibiae, which were the sites of the numerous old fractures, and the typical increased transradiancy and the very exuberant callus that may occur in this disease, were noted.

Figure XXI.

Central hydatid of the tibia. In this country one must always remember the possibility of hydatid disease. Note the tendency to extension along the line of least resistance, the medullary cavity.

Figure XXII.

Albers-Schönberg's disease in a female of the early twenties, from Royal Prince Alfred Hospital.

It has been said that the typical appearance of the skull will settle the diagnosis. However, though the skull picture may be definite, yet on the other hand I have again and again seen patients who have the changes typical of *osteitis deformans* with no skull changes or with very atypical skull changes; on the other hand in *osteitis fibrosa* it is said that skull changes occur (see Knaggs) which may approach the type of lesion seen in *osteitis deformans*. This is a condition I have not seen radiographically and I am still doubtful if one ever gets the woolly-like osteosclerosis typical of *osteitis deformans* in *osteitis fibrosa*.

THE DIAGNOSIS OF INTRACRANIAL LESIONS OF GENERAL INTEREST TO THE PROFESSION, REFERABLE TO DISEASES OF THE EAR.[1]

By A. W. Farmer, M.B., B.S. (Melbourne),
Honorary Surgeon to the Ear, Nose and Throat Department, Perth Hospital.

In bringing before you tonight the subject of the diagnosis of intracranial lesions referable to diseases of the ear, my aim was to choose one that would be of interest not to aurists only, but also to the physician, surgeon and general practitioner. I cannot claim any originality, the contents being merely a *pot-pourri* of information received from various clinics abroad.

Middle Ear Conditions.

Consider first purulent infections of the middle ear and let us follow the possible avenues by which infection can spread from here. For this purpose let us rapidly revise the anatomy of the middle ear which includes the Eustachian tube, tympanic cavity and its contents and the mastoid process. In considering the mastoid process, I shall consider the limit of the process, that is with the five groups of pneumatic cells fully developed.

The tympanic cavity is a four-sided cavity with a roof and a floor. It is divided into the attic or epitympanic space which lies above the floor of the *aditus* of the mastoid antrum. This process contains the head of the *malleus* and short process and body of the *incus* and merges into the *aditus*.

The lateral wall is bounded by the *membrana tympani*, the medial wall which because of its importance I shall consider separately. Anteriorly it converges into the entrance of the Eustachian tube, posteriorly it is bounded by the mastoid process. The roof is separated by thin bone from the middle fossa and the floor, by thin bone again from the jugular bulb.

On the medial wall of the tympanic cavity the following structures may be seen: (i) Facial nerve, (ii) geniculate ganglion, (iii) greater superficial petrosal nerve, (iv) *chorda tympani*, (v) lingual nerve, (vi) oval window leading into the vestibule of the labyrinth, (vii) foot plate of *stapes*, (viii) external semicircular canal lying on the floor of the *aditus*, (ix) round window also leading into the vestibule, (x) promontory behind which lies the basal turn of the cochlea.

It will thus be seen that a fistula through the oval or round window (rare) places the tympanic cavity in direct communication with the basal turn of the cochlea. Thus infection is carried into the labyrinth, setting up a purulent labyrinthitis and this infection, after having destroyed the labyrinth, passes on through the internal auditory meatus to reach the meninges, setting up a localized meningitis. When this happens, the meninges must be drained back by the way the infection entered and for this purpose the radical labyrinth operation is done. This consists in opening up the external semicircular canal at the *aditus*, the posterior canal in Troutman's triangle and the cochlea through the promontory, thus establishing free communication between the vestibule and its environs with the exterior. This is a life-saving measure in these conditions.

First of all one must consider how infection enters the middle ear. It enters in almost all cases by the Eustachian tube leading from the naso-pharynx. Why is it that in infants middle ear infections are so much more common than in adults? Alexander puts this most admirably:

1. The tympanic cavity in the infant is filled with mucus at birth which persists more or less during infancy. This is a good culture medium.
2. The Eustachian tube is shorter and wider than in the adult.
3. The prevalence of gastric disorders with vomiting rapidly infects the naso-pharynx.
4. The adenoid vegetations which become thus infected, are lying at the mouth of the Eustachian tube which rapidly transmits this infection to the middle ear.

Purulent Labyrinthitis.

I shall consider purulent labyrinthitis first, as I have already partially dealt with it and in considering it, will discuss only the diffuse purulent manifest type. As I said before, this may occur whenever a fistula exists from the infected middle ear into the labyrinth, be this secondary to a destructive process or trauma. The labyrinth becomes rapidly destroyed during this process, the patient complaining of severe vestibular symptoms, that is headache, violent vertigo and vomiting and spontaneous nystagmus to the opposite side. The functional tests on destruction of the labyrinth will reveal, of course, an absolute negativism, that is absolute deafness, no response to caloric or rotation tests and a negative fistula symptom—this of importance, if before death of the labyrinth a positive fistula symptom existed. By the fistula symptom is meant a conjugate deviation of the eyes on exerting pressure over the external auditory meatus. When a fistula exists between the middle ear and the labyrinth, this increased pressure in the tympanic cavity is transmitted directly through to the labyrinth.

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on March 25, 1928.

Subdural Abscess.

Subdural abscess occurs when infection spreads into the middle fossa. Subdural abscesses are divided into superficial and deep, the former when first overlying the roof, the latter when it spreads down along the petrous portion to the region of the internal auditory meatus and Gasserian ganglion. In the superficial there are no symptoms beyond headache. In the deep there are headaches, periorbital pain which is pathognomonic, sometimes even felt in the jaw and fifth and sixth nerve paralysis. In the same way we have superficial and deep subdural abscesses in the posterior fossa. Now, what follows if this process is allowed to go on, that is if you have pus lying between the dura and the bone? The dura naturally becomes granular and thickened, giving rise to a *pachymeningitis externa*, eventually the pus breaking, through with a *pachymeningitis interna* and circumscribed meningitis. Then the next stage is a localized encephalitis with spreading œdema and the formation of a brain abscess.

Meningitis.

When meningitis is confined to the middle fossa, it is known as a hood meningitis with no symptom beyond headache and the patients are frequently able to walk about. When involving the posterior fossa, it is known as a basal meningitis and this is the usual type which gives rise to the classical symptoms.

Headache is usually localized more or less, but may be very general. It is very much exaggerated by any pressure on the head or the slightest movement. The character of the headache is most important in differentiating it from that of a cerebral abscess. In the former the patient rises with a meningeal cry which gradually subsides, whereas in the latter the cry commences as more of a groan working up in a *crescendo*.

General spasticity, particularly of the neck, is present with a Kernig's and a Bondi's sign (turn the patient's head to one side and the leg from the side turned comes up with the movement of the head).

Nystagmus is present usually to either side and is of no value.

In regard to lumbar puncture, the old maxim about never puncturing in a case of an intracranial complication because of the risk of rupturing a cerebral abscess, is now dead. There is no danger whatever in slowly withdrawing two to five cubic centimetres for diagnostic purposes and most valuable information can be so gained.

In a meningitis the fluid is, of course, as in other brain lesions, under increased pressure. It is cloudy or purulent, depending on the extent of diffusion, contains polymorphonuclear leucocytes and on culture mostly streptococci and diplococci. In a tuberculous meningitis, on the other hand, the fluid is clear or opalescent, at the most contains mononuclear leucocytes instead of polymorphonuclear and is usually sterile, although tubercle bacilli may be obtained. In a brain abscess which I shall refer to here for differential diagnostic purposes, the fluid is clear, cloudy or purulent, contains polymorphonuclear leucocytes in abundance, sometimes a few mononuclear leucocytes, but no bacteria.

Temporal Lobe Abscess.

Temporal lobe abscess may exist for many months unknown to the patient or his surgeon. After reaching the brain, infection spreads along the cerebral vessels or in the form of a spreading thrombosis of the central artery. These cerebral arteries are end arteries, hence local abscess with spreading necrosis. How does this occur? When this infection commences to spread along the vessels, an œdema is produced with resultant cerebral compression. Now the skull is a practically closed cavity the only contents that can escape from it being blood and cerebro-spinal fluid. Now when this œdema forms around the spreading infection, something has to make way inside the skull and it is the cerebro-spinal fluid which is forced out with resultant increase of cerebro-spinal fluid pressure with formation of more œdema and thus a viscious cycle is set up. The fluid also contains metabolites which act as irritants to the cortex directly and particularly to the medullary centres, with consequent rise of blood pressure. Finally, the local pressure is sufficient to occlude the arteries completely with an area of necrosis and commencing abscess.

Ultimately this abscess reaches the lateral ventricle into which it ruptures with the formation of a rapidly fatal meningitis. This cycle, however, may become arrested for a considerable period, thus we can distinguish two stages: (i) A manifest stage, that is during the spread in which signs and symptoms will be present and which may only last a few hours. (ii) A latent stage or stage of arrest which may last for several months with no symptoms beyond some degree of headache. There is a perfectly authentic case of a professional footballer who was seen by an outside doctor in Vienna with all the typical signs of a manifest temporo-sphenoidal abscess. The doctor immediately sent him to the clinic for admission and when he was seen there two hours later there were no definite signs at all. After being retained under observation for some weeks, he was discharged and played football regularly for some months. Ultimately he died suddenly during another manifest stage in which rupture of the abscess occurred into the lateral ventricle.

Symptoms of the Manifest Stage.

The symptoms of the manifest stage are as follows:

The patient is drowsy and difficult to rouse and if he can be roused, does not take any interest in what you are saying, but only complains of headache. If he wants to eat, he eats voraciously; if he does not want to, nothing on earth will induce him to.

In regard to headache, the type has already been discussed under meningitis and it is usually local-

ized to the neighbourhood of the temporo-parietal lobe.

Tenderness on percussion is sometimes present in this area.

The temperature is usually about 37·7° C. (100° F.) with gradually slowing pulse.

Nystagmus very rarely occurs. When the abscess ruptures into the ventricle, the typical vertical nystagmus is seen, always of grave prognosis, and is due to irritation of some unknown centre in the medulla. Deiter's nucleus, the centre for nystagmus, has apparently nothing to do with this vertical nystagmus.

Choked disc only very rarely occurs.

Now let us stop to consider the centres in the temporal lobe. In Hesch's gyrus, in the temporal lobe is situated the centre for hearing and around that is the Wernicke centre. In the occipital lobe is the centre for sight. In the frontal lobe is the Broca or word-speaking centre. All these centres are linked up with each other and with the cortex by association fibres.

The Wernicke centre is the storehouse of memorized words, seen or heard. For instance, take a word of which you have forgotten the meaning. You cannot think, on hearing the word, what it means. You are then shown the object and its meaning is immediately recalled. Thus you see the necessity of these association fibres passing between the two centres of hearing and sight, to release the word from the Wernicke centre. Now, when these association fibres become involved by a spreading œdema around a cerebral abscess, the Wernicke centre is thrown out of action. How does this show itself? By the patient, when shown an object, being unable to name it but describing its meaning or its use. For this the Wernicke centre is not required. This condition is known as anomia.

Another condition is paraphasia, that is the calling of every object shown by the one name (perseveration). This is a sensory aphasia. This centre of language is situated in the left brain for right-handed people and *rice versa*. When the centre on the left side becomes destroyed, the right-sided centre which is in abeyance, soon becomes developed and compensates in a matter of two or three weeks.

There is one other important sign present on either side of the brain and fairly constant, that is a partial hemianopia on the same side as the lesion.

If you take a frontal section through the temporal lobe, you will notice that the inferior horn of the lateral ventricle is in very close relationship to the optic nerve fibres passing from the optic chiasma. An abscess of the temporal lobe always tends to spread towards this inferior horn and thus these fibres become pressed upon, giving rise to this hemianopia on the same side as the lesion.

Sinus Thrombosis.

So far I have dealt only with conditions tracking through the tympanic cavity. Now I shall come to gross abscess formation in the mastoid cells and follow the sequelæ.

The first condition one meets with, as a rule, is a perisinal abscess, that is a breaking down of the cells with abscess formation around the lateral sinus.

Sooner or later the sinus wall becomes infected as shown by the presence of granulations on the wall, ultimately a thrombosis forms in the sinus. This is at first mural or partial, gradually increasing in size until it becomes occluding, then completely blocking the sinus. This thrombosis may spread in both directions backwards towards the occiput to the torcular or junction of the lateral sinus with that of the other side and the superior longitudinal sinus. The other direction of spread is forward to the jugular bulb and thence down along the internal jugular vein. The great danger associated with sinus thrombosis apart from local absorption, is the formation of metastases and, therefore, the imperative need for immediately tying the jugular vein on the discovery of a thrombosis is seen.

The symptoms of sinus thrombosis are:

1. The patient is euphoric, that is though very ill indeed, he feels remarkably well.

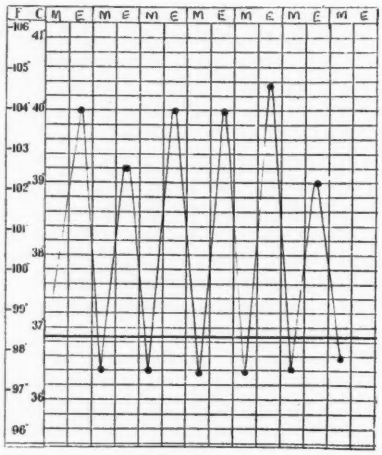

Chart I.

2. Temperature is grossly septic, as shown diagramatically in the acompanying chart (Chart 1), the important feature of the chart being that the morning temperature is subnormal and there are usually present frequent and repeated rigors. In a simple toxæmia you get a chart as seen in Chart II, where it will be seen that the morning temperature is raised above the normal. In a mixed case, where both toxæmia and in addition bacteria are in the blood stream, you get a combination of the two charts as shown in Chart III.

3. Hæmatogenous icterus is seen under the conjunctivæ.

4. Petechiæ are seen under the skin and epistaxis occurs.

5. Choked disc according to Ruttin is of great importance, but appears rather late to be of value, about the sixteenth to thirtieth day after occlusion

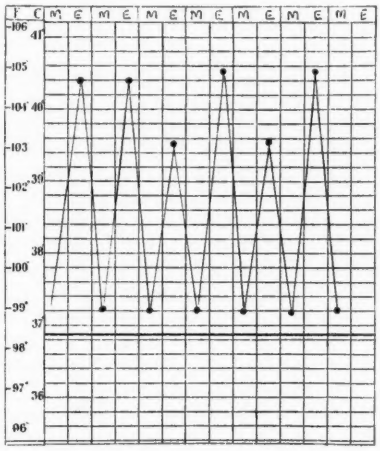

Chart II.

of the bulb. Ruttin maintains that it is due to a stasis in the cavernous sinus.

6. Metastases occur, mostly in the lung, but frequently also in the liver and kidneys. The later the metastasis developes, the better the prognosis and these late metastases usually have special sites, namely, Douglas's pouch, the perineum and the eyeball. The serious early ones occur usually in the subcutaneous tissue and muscles and are frequently multiple and of very grave prognosis.

Cerebellar Abscess.

Cerebellar abscess occurs in 90% of infections associated with labyrinthine disease (Ruttin), also by direct necrosis into the posterior fossa secondary to a large and destructive perisinal abscess. I am rather inclined from reading the literature to think that Ruttin places his figure unduly high and this latter focus accounts for more than the remaining 10%.

1. The diagnosis of a cerebellar abscess with a live labyrinth is very difficult, with a dead one it is much easier. It is the nystagmus which is so valuable in the diagnosis. The type of nystagmus varies in the two conditions. Labyrinthine nystagmus is a fine nystagmus always to the opposite side of the lesion (that is speaking of a suppurative labyrinth). The nystagmus due to a cerebellar abscess is much coarser and may be to either side, but more usually to the same side as the lesion. Also the nystagmus in a labyrinthine lesion tends to diminish after the labyrinth is destroyed; in a cerebellar abscess it increases as the pressure becomes greater.

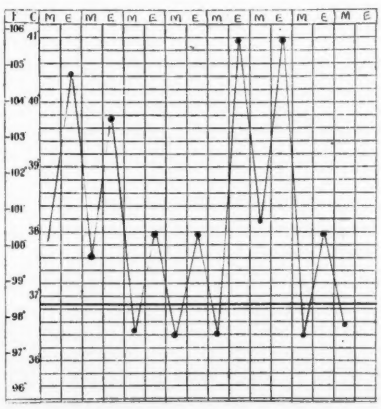

Chart III.

2. Choked disc is more common than in a temporal lobe abscess.

3. Vomiting is constant as a rule.

4. Headache is constant and may be either occipital or frontal.

5. Vertigo is definite. The patient tends to fall to the opposite side of the lesion. Here again the direct opposite to a labyrinthine affection. If situated in the *vermis* the patient always tends to fall backwards, the reason of which is not explainable. The direction of the past-pointing follows the direction of falling.

6. Disdiadokokinesis is fairly constant.

7. Loss of sense of shape of objects through pressure on the supramarginal lobe is noticeable.

The prognosis of temporal abscess and of cerebellar abscess is rather interesting. In Vienna Fremmyl stated that since 1920 at the Neumann clinic, which is the largest ear clinic in the world, not a single patient with cerebellar abscess has recovered, whereas in temporal lobe abscess cases a 40% recovery is claimed. Ruttin, at the Rudolf Spital, on the other hand, claims 5% recovery in cerebellar and 15% recovery in temporal abscesses.

McEwan, who did the pioneering work on this subject, claimed an 80% recovery, but did not differentiate between cerebral and cerebellar abscesses, however.

Acoustic Nerve Tumours.

So far I have dealt only with the spread of sepsis from the middle ear. There is, however, one other interesting subject which has received greater prominence lately, mainly through the work of Harvey Cushing, and this is acoustic nerve tumours. Their interest to the profession at large is that their presence ultimately causes pressure in the cerebello-pontine angle with the usual train of signs presenting in sequence or rather they should do so.

These tumours are pathologically really neurofibromata. They arise at the external margin of the internal auditory canal as a rule with a very small area of attachment. They gradually erode the internal auditory canal and grow out into the cerebello-pontine angle, frequently covered by an arachnoid cyst. The earliest signs are, of course, referable to the eighth nerve, namely, deafness, tinnitus and vertigo which become progressive, together with facial paresis as the seventh nerve accompanies the eighth in the canal. Later, as the tumour enlarges the fifth and sixth nerves become involved and still later the ninth, tenth and eleventh. The *chorda tympani* owing to its forma-

tion in the seventh nerve consequently becomes early affected by paresis with the loss of taste over its distribution on the tongue.

At the annual congress of the British Medical Association at Edinburgh last year some very interesting radiographs were shown by Dr. H. Graham Hodgson. In these radiograms he demonstrated that these tumours could be diagnosed at an early stage by a greatly widening and increasing erosion of the internal auditory meatus. This excellent work of Dr. Hodgson will mean that by the early diagnosis of these tumours surgical treatment will be resorted to long before serious damage is done to the surrounding nerves and other structures, particularly as the progress and the appearance of signs is so extraordinarily slow that naturally diagnosis is very difficult.

Acknowledgements.

I have to thank the following gentlemen chiefly for the subject matter of this paper: Dr. J. S. Fraser, of Edinburgh, Mr. Watkyn Thomas and Dr. Graham Hodgson, of London; and Professor Ruttin, Professor Alexander and Dr. Fremmyl, of Vienna.

SOME ANTE-NATAL OBSERVATIONS ON TWO THOUSAND CASES.[1]

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Melbourne.

IN this short paper I have endeavoured to deal with ante-natal work from the standpoint of the man engaged in general private practice, so I shall not make it an opportunity of airing my text book knowledge or of quoting hospital statistics, but shall try to give you the results of my personal observation during the past fifteen years in a series of two thousand patients in private midwifery practice.

I know of no branch of medicine in which originality is so much discouraged as in obstetrics. The deplorable dulness and the painful scientific expositions of midwifery text books are, I am sure, largely responsible for the light esteem in which the work is held. All honour to science; yet I feel instinctively that one can be perhaps too scientific in dealing with a human being especially a woman.

The future development of obstetrics work must be along ante-natal lines. Perfect ante-natal care means a simple and safe delivery for both mother and child; that is the aim of all obstetric practice. Of course, I must admit that ante-natal work is pure philanthropy. Patients do not, many cannot afford to pay for it. To do one's best work regardless of monetary reward is the inevitable destiny of the faithful obstetrician. We may take comfort from the fact that it is just this attitude towards any work which distinguishes the artist from the tradesman.

[1] Read at a meeting of the Gynæcological and Obstetrical Section of the Victorian Branch of the British Medical Association, March, 1927.

Medicine today has become commercialized; this fact also contributes towards the definite unpopularity of obstetric work.

During the first few years of my practice, my patients rarely engaged me before the sixth month, usually later. Now I rarely make an engagement with a patient later than the third month of pregnancy, the tendency being for women to avail themselves of ante-natal care from the sixth to the tenth week. Of course, I have always emphasized the importance of care in the early months, but there is also now a general recognition of the necessity for ante-natal care. Management of pregnancy entails consideration of the whole female economy quite apart from the uterus and its contents. For that reason, therefore, the best obstetrician is of necessity the sound general practitioner, with a special interest in midwifery. A great deal can be learnt about a patient by general observation before an examination is made.

One should be able to tell by her walk and attitude the period of gestation fairly accurately. If a patient shows no definite tendency to lean backwards in the early months, the pelvis can well accommodate the growing fœtus and should offer no obstruction to delivery. The general build of a woman will give some indication of the pelvic measurements and of the type of pelvis to expect. The muscular system can be judged; a healthy athletic woman should possess uterine muscle capable of overcoming some degree of obstruction during delivery. In the later months the shape and projection of the abdomen quickly show any contraction or abnormality of the pelvic brim. By a patient's general appearance—her eyes, the colour of her skin, the condition of her hair—one can estimate her nutrition, her digestive capacity, the presence or absence of constipation, also the hygienic conditions under which she lives. The condition of her clothing is an indication of the cleanliness or otherwise of the patient's habits. The presence of anæmia can be detected, also an estimate of the patient's nervous condition can be made by observing her speech and general behaviour. By a certain strained expression I have learned to recognize those patients in whom pregnancy is causing nervous exhaustion. Excessive tiredness and languid movements and a certain muddy complexion suggest glandular insufficiency. There is a type of face I always associate with renal insufficiency. The skin is pale and anæmic, the eyes look heavy and there is often some puffiness of the skin under the eyes.

General Examination.

Before considering the pregnancy at all I make an exhaustive general examination of the patient.

The family history is of interest and should be inquired into for the following: (i) Nervous disorders—insanity and epilepsy, (ii) hæmophilia, (iii) tendency to tuberculosis, (iv) multiple pregnancy, (v) position in family. The last member of a large family or one born at a short interval after the preceding member often manifests glandular

insufficiencies. (vi) The age of parents at time of the patient's birth. A child of middle-aged parents often suffers from nervous disorders and glandular disturbances.

I inquire into the patient's general health, habits and hygiene and past illnesses with special reference to scarlet fever and rheumatic fever.

I then make a careful general examination of: (i) The mouth, teeth and tonsils, (ii) the digestive system, (iii) the heart, (iv) the respiratory system. I note any old pleurisy or a suspicious apex, as an irregular temperature curve in the puerperium may be the only symptom of a pleural effusion or other active tuberculous lesion. I examine (v) the blood vascular system, test for anæmia or syphilis. Blood pressure is most important. A low blood pressure may indicate glandular treatment, a high blood pressure may be the early sign of an unsuspected toxæmia. The presence of varicose veins should be noted. (vi) The renal system. I examine the urine as early as possible; most of my patients bring a specimen when they come to engage me, especially *multiparæ*. (vii) The bladder is taken into consideration. A suspicious increased frequency is often the only symptom of cystitis and early treatment is essential. Lastly (viii) the breasts demand attention. I notice the size, shape and condition of the nipples.

Special Examination.

If the patient is a *multipara* I go into the previous history of pregnancies, confinements or miscarriages. I consider the general appearance of the abdomen and decide whether the height of the fundus corresponds with the period of gestation given by the patient. If any operative scars are present, I inquire into the nature of the operation, in case of the possibility of uterine adhesions.

Pronounced striæ I find only in those few patients who engage me late in their pregnancy. Early abdominal massage and proper abdominal support from the fourth month onwards lessen, if not entirely obliterate, these marks.

Pelvimetry.

I measure the intercristal and interspinous and intertrochanteric lines and record any deviation from normal, 27·5 centimetres (eleven inches), 25 centimetres (ten inches) and 35 centimetres (fourteen inches). I note the relationship of the trochanters to the iliac crests. The patient is then put in the Sims position. I measure the distance between the ischial tuberosities and estimate the same manually.

Internal Pelvic Examination.

Vaginal discharge is always increased during pregnancy. The character of the discharge and the presence of irritation of the labia and the mucous membrane near the meatus will indicate any infection and the necessity for further investigation. The condition of the vaginal walls should be examined for signs of old infection, the roughening left by gonorrhœal infection (nutmeg grater). I examine the cervix for scars in a *multipara* and erosions in both *primiparæ* and *multiparæ*. If I find a large tear in a *multipara*, I note it and warn the patient to be very careful during the early months as I consider severe cervical lacerations a frequent cause of abortion. Should the patient abort, I repair the cervix before allowing her to go through another pregnancy.

I then measure the distance digitally from the sacral promontory to the pubic arch. I sweep my forefinger round the brim on both sides and feel from the sacral promontory and tip of the coccyx for any bony excrescence and also to determine the curve of the sacrum and the depth of the pelvis. I decide whether the coccyx projects forward so far as to lessen the antero-posterior diameter of the outlet (antero-posterior diameter 12·5 centimetres or five inches). The angle formed the pubic arch can be estimated and the internal distance between the ischial tuberosities (normal ten centimetres or four inches).

I examine the uterus bimanually to compare the size of the fœtus and the size of the brim and also to determine the period of gestation. If the uterus is larger than the period estimated by the patient herself, I inquire into the history of the last menstruation. If anything unusual was noted about the time or the amount of flow, I ante-date the pregnancy by one month. If not, I bear in mind the following contingencies: (i) Unusually large fœtus, (ii) multiple pregnancy, (ii) hydramnios, (iv) myomatous uterus and I ask the patient to present herself for reexmination at the seventh month.

If the uterus is retroverted and freely movable, I advise postural treatment in the early months. If not freely movable, I watch the patient for signs of incarceration; if necessary a ring pessary is used until the uterus is too large to reenter the pelvis.

If I find a tendency to prolapse, I advise absolute rest for the first three or four months or until the uterus becomes an abdominal organ. When this is not possible, I insert a ring pessary, but this treatment has not given me encouraging results.

General Treatment.

I supply each patient with a card of general instructions and a list of symptoms which should be reported.

Diet.

Diet should be plentiful, varied and well balanced, but the patient's needs vary according to the period of gestation.

From the first to the fourth month the diet should be rich in easily digested carbohydrate food. From the third to the eighth month the diet must supply an excess of calcium; if milk seems insufficient or disturbing to the digestion, calcium must be supplied in the form of drugs. From the eighth month onward carbohydrate must be limited, sugar and sweets being specially avoided. It is essential

to supply iron at this period. The iron content of milk, both cow's and human, is low and insufficient in itself to supply the needs of the growing child, so that the child is largely dependent on the iron stored in the liver at birth. Most of this iron store is laid down in the later months of pregnancy, so that the premature infant born with a small iron deposit invariably becomes anæmic, often profoundly so. Underdone red meat once a day at least should be eaten; if any tendency to anæmia is present, iron in drug form must be administered. Throughout the period of pregnancy fresh fruit and vegetables must be eaten freely. In patients whose weight increases rapidly, I limit the carbohydrate and fluid intake as far as possible and if necessary administer small doses of thyroidin.

A regular aperient is very necessary,, the drug varying with the patient's constitution and inclination.

Whenever there is a history of hæmophilia or previous *post partum* hæmorrhage I feed patients on calcium and iron.

Sleep.

I like my patients to sleep outside if possible; in any case I insist on thorough ventilation of the sleeping room. I also urge them to sleep for an hour in the afternoons.

Baths.

I advise a daily bath (hot) as much for its effects on the nervous system as to insure cleanliness.

Care of Nipples.

When a patient engages me, I ask her to wash her nipples daily with soap and warm water and to dry them thoroughly, rubbing well with a towel and from the seventh month onwards to swab the nipples with equal parts of olive oil and spirit.

Dress.

Modern dress is so sensible that it meets all the requirements of the pregnant woman, but I advise against high heeled shoes, as they upset the natural balance of the body during pregnancy. After the fourth month I insist on the wearing of a properly fitting maternity belt or corset. Attention to this matter adds considerably to the patient's comfort. It not only prevents undue stretching of the abdominal muscles and the formation of striæ, but gives the patient a feeling of well being and a capacity for free movement she may otherwise lack. Proper support also prevents most of the pains and cramps of the over-stretched muscles so common in the later months of pregnancy. Even after the puerperium I never allow a patient to get up until she has been fitted with a suitable corset.

Half the troubles of multiparous women are, I am convinced, due to insufficient or unsuitable abdominal support.

Minor Complications of Pregnancy.

I shall now consider the commoner minor complications of pregnancy. In treating all these conditions, I bear in mind that the nervous system of the pregnant woman is less stable than that of the normal woman and responds in an exaggerated manner to any abnormal condition either local or general, however slight. I find that the particular treatment necessary for any complication which may arise, combined with a sedative mixture, gives infinitely better results than the same treatment without sedatives. The nervous system should be spared in every possible way in daily life and all treatment should have the one object of building up the patient's nervous reserve to meet the tremendous emergency of parturition.

The Digestive System.

Of all conditions connected with the digestive system I think heartburn is most common. I rarely see a patient who does not complain of this symptom. It is quickly relieved by increasing the carbohydrate and giving the following mixture every four hours:

℞ *Sodii bicarbonatis* 1·0 gramme or 15 grains.
Ammonii bromidi 0·66 gramme or 11 grains.
Tincturæ nucis vomicæ 0·6 mil or 10 minims.
Infusum gentianæ compositum ad 15·0 mil or half a fluid ounce.

In mild cases bicarbonate of soda gives relief.

Constipation.

I regard constipation as the most serious of the minor complications of pregnancy. It is the forerunner of all the serious toxæmias as well as of hæmorrhoids. Bowel toxæmia I consider so important that in examining all specimens of urine I first test with cold nitric acid for the indican ring. Where this is present, I take pains to correct by a purge and daily aperient, keeping the patient under close observation until this reaction disappears. Where there is constipation, together with very painful hæmorrhoids, I send the patient into hospital for two or three days, give a sedative and have the bowel thoroughly emptied by a high enema, afterwards treating the hæmorrhoids and constipation.

Morning Sickness.

Morning sickness usually begins in the sixth week and continues to the thirteenth or fourteenth week. The vomiting occurs after the longest fasting period when the liver is depleted of its glycogen, hence the necessity of increasing the carbohydrate intake during the early months. Nervous exhaustion, following any severe nervous strain, both in male and female patients, frequently causes nausea and vomiting in the forenoon. The morning sickness of pregnancy and of nervous exhaustion are strikingly similar.

In pregnancy there is a tremendous stimulus to all glandular functions and it probably takes time, perhaps six to eight weeks, for a normal balance to be reestablished.

Does the dissipation of nervous energies in abnormal directions, such as pregnancy, or nerve stress disturb the glandular balance and so alter metabolism?

Pregnancy is, of course, a continuous strain, whereas other strains may be only temporary. Displacements of the uterus may be a contributing factor in morning sickness, but I do not attach much importance to them as a definite cause. I treat morning sickness at first by regulating the diet; if this is ineffective, I give the mixture mentioned before, an alkaline gentian mixture with strychnine and bromide of ammonia, and only very rarely have had to treat a patient further.

If the vomiting begins to distress the patient, I put her into hospital and exclude her husband and family. It is generally found that some irritating factor in her surroundings causes the vomiting; unfortunately it is often the husband and all one's powers of suggestion have to be exerted to overcome the difficulty. One of my patients ceased vomiting on admission to hospital, but vomited violently for hours each time her husband was allowed to see her. In this case in spite of treatment she became worse and I had to empty the uterus. On only two occasions I have had to empty the uterus to stop vomiting. In my experience *hyperemesis gravidarum* is a very rare condition.

The glucose excreted in diabetes may exceed the intake of carbohydrate, the demands of the rapidly growing fœtus may produce the same result in a pregnant woman, but during the last few years I have not met with a case of *hyperemesis gravidarum* and have not had the opportunity of testing treatment with "Insulin" and glucose.

Palpitation.

Palpitation, apart from any cardiac lesion, is a very common symptom of pregnant women. It is a sign of calcium deficiency and responds readily to treatment by calcium lactate or "Tricalcine." The patient takes the latter fasting in the morning. I give calcium lactate, one gramme (fifteen grains), three times a day until symptoms disappear. If it recurs, the same treatment is continued.

Pruritus Vulvæ.

Pruritus vulvæ is a common and distressing symptom, occurring after the fifth or sixth month. In my experience it is usually associated with internal hæmorrhoids, although I do see patients in whom these are apparently not present. The association of vaginal discharge is mentioned later.

Besides treating the hæmorrhoids, I give a sedative mixture.

℞ *Phenazoni* 0·54 gramme or 11 grains.
Sodii bromidi 0·48 gramme or 8 grains.
Tincturæ gelsemii 0·36 mil or 6 minims.
Syrupi aurantii 2·0 mil or half a fluid drachm
Aquam chloroformi ad 15·0 mil or half a fluid ounce.

This gives excellent results, given three times a day for two days, then twice daily, morning and evening or only once late in the day, as it is always at night that this symptom causes most discomfort.

Another unpleasant symptom, complained of in the later months, is an irritating vaginal discharge, non-specific and non-purulent. It is acid in reaction and may cause pruritus. An alkaline douche and any ointment with a petroleum base, applied very freely to the vulva, relieve the soreness and irritation, but a sedative mixture may have to be given.

Hydrorrhœa Gravidarum

Hydrorrhœa gravidarum is according to text books a rare condition, but I have met with several cases in the last four years. The discharge is intermittent, usually coloured, but the colour is paler than that of the usual show and is less viscid than the other vaginal discharges. In all these cases the membranes were friable and abnormal in appearance and in one instance the placenta was completely adherent and most unhealthy in appearance. The condition is of interest, as the patient often thinks she had had a show and a differential diagnosis has to be made between hydrorrhœa, imminent labour and slight *ante partum* hæmorrhage. When the discharge is colourless and more copious, the diagnosis lies between ruptured membranes and hydrorrhœa. Brownish discharge due to a dead fœtus with its signs and symptoms is easily recognized.

Cramps.

Since I have paid more serious attention to effective abdominal support, my patients rarely complain of cramps. Muscle cramps may be due to two causes: (i) Overactivity or overstretching of the muscles concerned, (ii) a definite toxæmia; wherever this symptom persists, I regard it as an indication for further investigation of the excretory functions and the elimination of any possible form of infection.

I find varicose veins are now a less frequent complication of pregnancy than in the earlier years of my practice. This again is accounted for by women availing themselves of ante-natal care in the early months. Whenever I find any pronounced tendency to varicosity, I put the patient to bed for a fortnight; afterwards, with a reasonable amount of rest, abdominal support and, if necessary, crepe bandages, the patient carries on quite comfortably. I advocate a three years' interval before a subsequent pregnancy; the veins then give no trouble.

Ante Partum Hæmorrhage.

Ante partum hæmorrhage in the first three months may be (i) a painless hæmorrhage, slight in amount; it may occur at the time of the usual menstrual flow. It may be (ii) hæmorrhage more or less severe and accompanied by pain due to threatened or inevitable abortion.

In the first of the two varieties no treatment is required beyond rest and I need not go into the treatment of the second type.

From the third to the sixth month hæmorrhage means threatened or inevitable miscarriage. From the sixth month onward the hæmorrhage may be due to (i) onset of early labour, (ii) *placenta prævia*, (iii) accidental hæmorrhage.

With regard to *placenta prævia* I adopt conservative measures wherever possible; but if the

patient is too far away to remain under skilled observation, I terminate the pregnancy.

I consider accidental hæmorrhage of far more frequent occurrence than text books would lead one to suppose. Many hæmorrhages are slight, the danger then being greater to the fœtus than to the mother. In some instances there may be a little *ante partum* bleeding, but otherwise the course of labour is normal, resulting in the birth of a healthy child; but the placenta is followed by old blood clot and may contain an area of organizing clot.

In hæmorrhage involving a larger area of separation of the placenta the child, if born alive, is in a state of asphyxia or may be still-born (sometimes in a state of *rigor mortis*). The mother is apparently unaffected and I have not noticed any tendency to *post partum* hæmorrhage. I need not discuss the treatment for the classic type of accidental hæmorrhage with complete separation of the placenta.

Toxæmia.

Albuminurics I divide roughly into three classes:

1. Those in whom albuminuria developes suddenly any time after the sixth month—usually eclamptics.

2. Those with albumin from the beginning or from the third month—those with true renal lesions.

3. Those developing œdema and other pressure symptoms together with albuminuria late in pregnancy, the albumin disappearing after parturition.

I have found albumin in the urine of all patients I have had the opportunity of examining just before the onset of labour. This is especially the case with multiple pregnancies and a large fœtus, but it is not accompanied by symptoms of toxæmia.

In the first class I put the patient to bed, give mild diuretics (alkaline) and suitable diet, regular aperients. I watch the blood pressure and if the patient's condition is not improving, I terminate the pregnancy, waiting if possible until the child is viable, if the condition occurs soon after the sixth month.

I have sometimes seen women with nervous symptoms, such as eye-movements resembling nystagmus and general restlessness, with a high blood pressure, but with little or no albuminuria. I regard them as threatened eclamptics. In two cases in which my advice was not taken, the patient later developed convulsions.

In the second class of albuminurics the question is mainly one of prognosis, especially when the patient is a *multipara* and has one or more children to be considered. If the albumin is present at the second or third month and the patient gives a history of previous complicated pregnancy or if she is a *primipara* with a history of scarlet fever or nephritis, I do a quantitative test of the albumin, have the eyes examined and a blood urea estimation made. If pregnancy is contraindicated, I have no hesitation in terminating it. If, however, there is a possibility of allowing it to continue until the child is viable, the treatment is mainly a question of sufficient rest, diet and aperients. I limit the protein and allow meat only once a day, preferably red—either mutton or lamb. The question of salt-free diet must be considered; one authority prescribes one salt-free week in every four as a prophylactic. A liberal allowance of fruit and vegetables (except tomatoes and rhubarb) must be made, together with brown bread, milk and any cereal food. Here, of course, the economic factor largely influences the treatment. If the woman cannot afford the rest required, she cannot afford the pregnancy. These patients must be kept under the strictest observation for further signs of failing renal function and development of any cardio-vascular or respiratory complication.

In both these classes should a convulsion occur or should labour begin, I administer morphine and scapolamine in the former combined with the usual eliminative treatment necessary. I find that in the last three or four years various Continental authorities have advocated this form of narcosis as a prophylactic against convulsions in threatened eclampsia.

In the third class, if rest and treatment have no effect on the œdema, I induce labour.

Cystitis.

Cystitis is very common and is usually due to mechanical pressure of the uterus, especially in the early months. In my experience almost every specimen of urine tested in the first four months shows signs of mechanical irritation of the mucous membrane of the bladder, such as shreds, mucus and cells. Cystitis also occurs frequently in conjunction with a definite or potential cystocele which increases the amount of residual urine. The condition, if treated early, responds quickly to the usual routine of rest, diet and an alkaline mixture containing some antiseptic such as citrate of potash combined with "Hexostate." If there is much pain, I find that bromide of soda and hyoscyamine give great relief. The cystitis may recur during the pregnancy, so the patient must be kept under observation and frequent urine examinations must be made.

This tendency to cystitis should be remembered, as it so often recurs in an intensified and more obstinate form after parturition; it may also then be accompanied by the worst of comrades, pyelitis.

Pyelitis.

I now come to pyelitis—the *bête noire* of all obstetricians. I know of no condition through which one loses more peace of mind than pyelitis. This complication usually developes in the fifth month; the onset may be acute and the diagnosis extremely difficult between pyelitis and appendicitis without exhaustive urinary examination.

Fortunately all the pyelitic infections of pregnancy which I have encountered have been of the mild variety and have cleared up under the usual

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

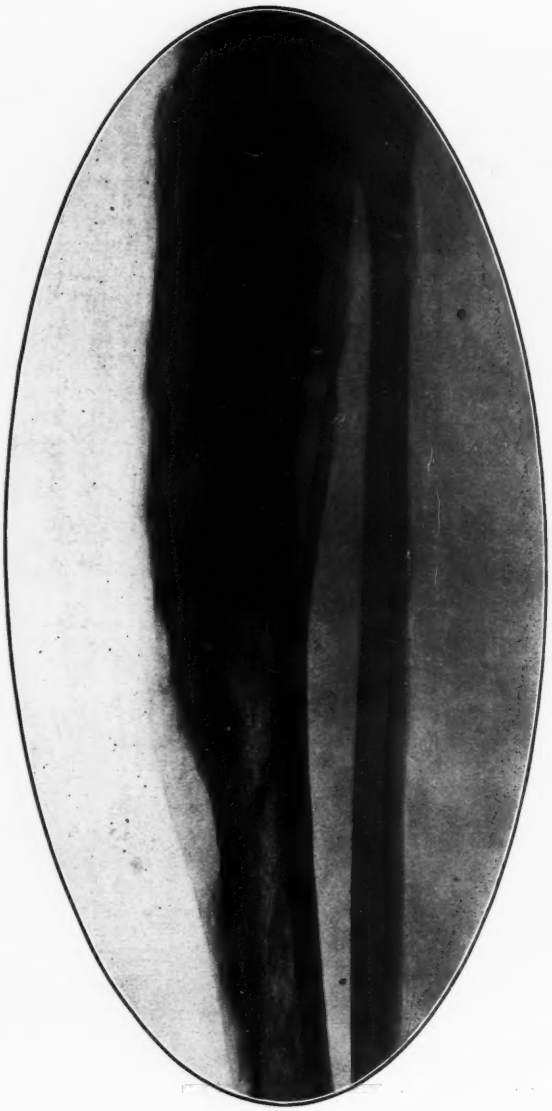

FIGURE II.

FIGURE III.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE IV.

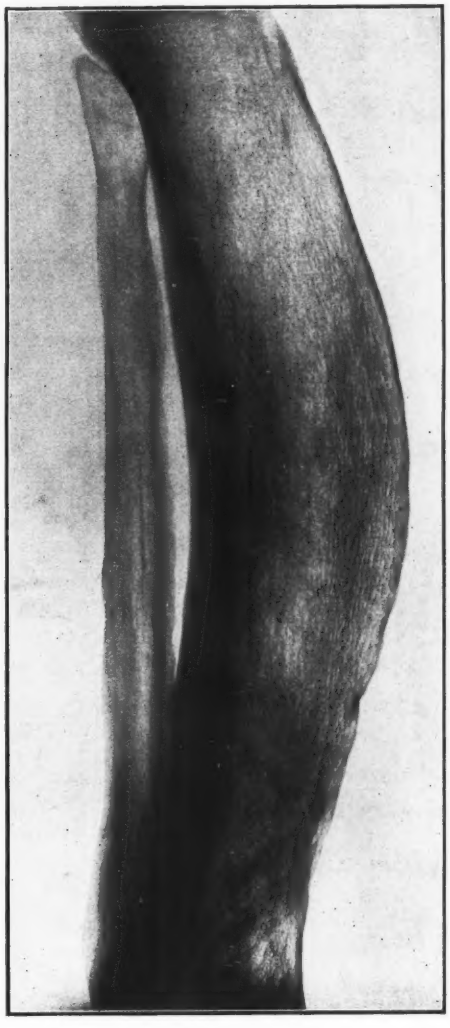

FIGURE V.

ILLUSTRATIONS TO THE ARTICLE BY Dr. H. R. SEAR.

Figure VI.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE VII.

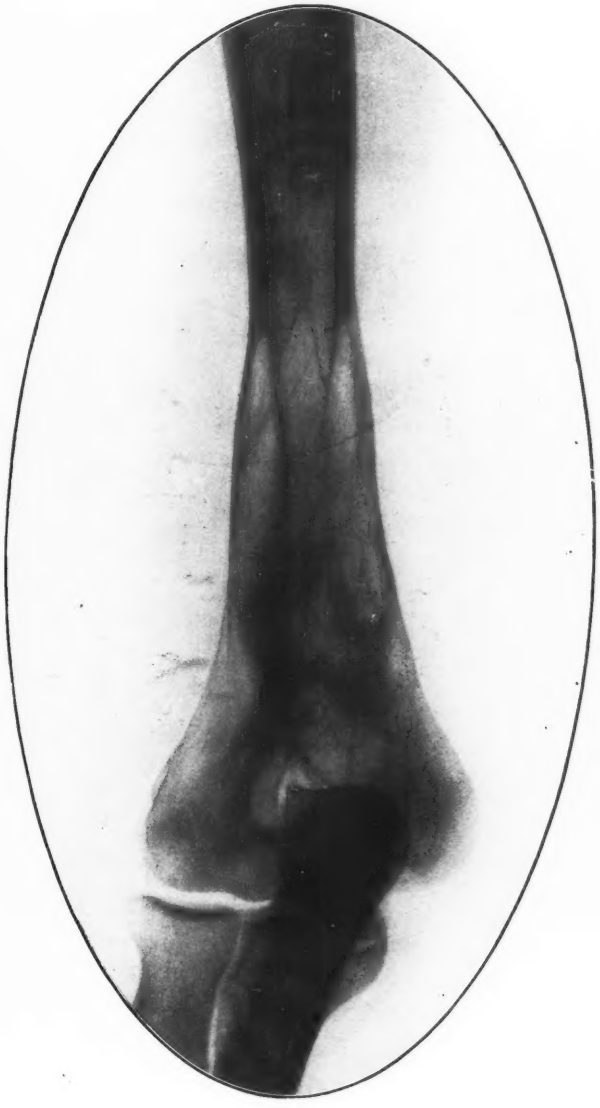

FIGURE VIII.

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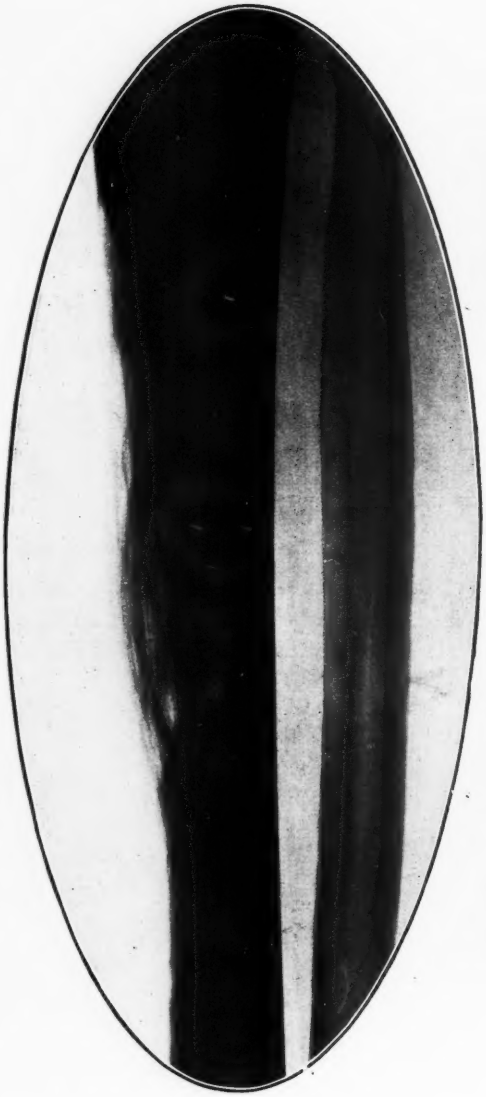

FIGURE IX.

FIGURE X.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE XI.

FIGURE XII.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE XIII.

FIGURE XIV.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE XV.

FIGURE XVI.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

FIGURE XVII.

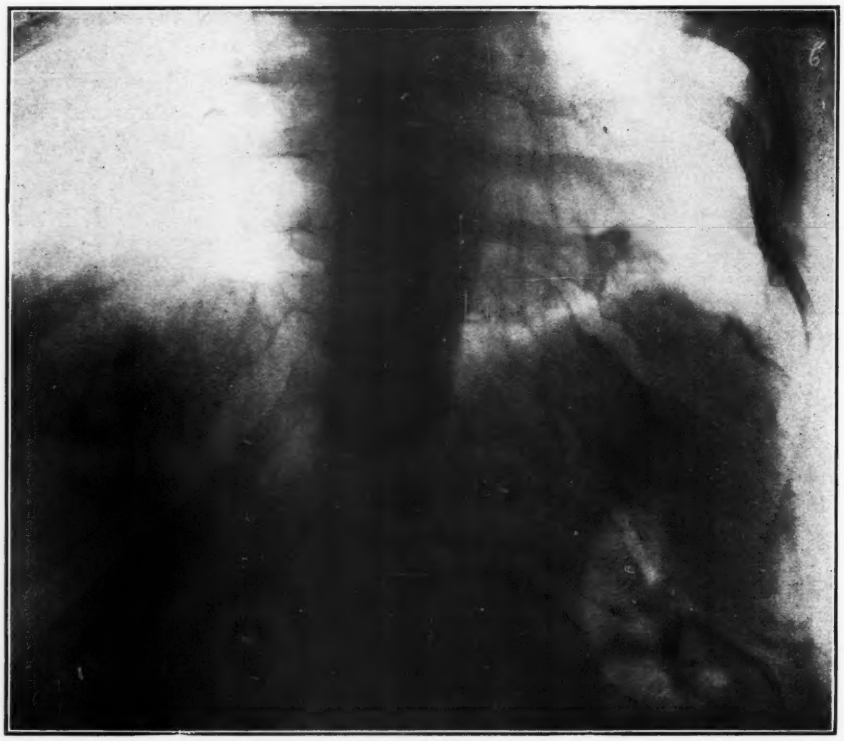

FIGURE XVIII.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

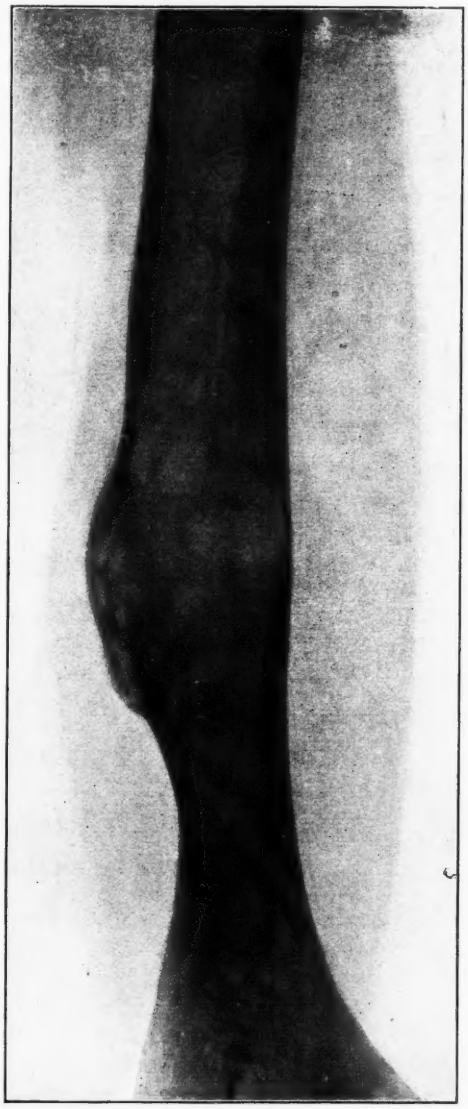

FIGURE XIX.

FIGURE XX.

ILLUSTRATIONS TO THE ARTICLE BY DR. H. R. SEAR.

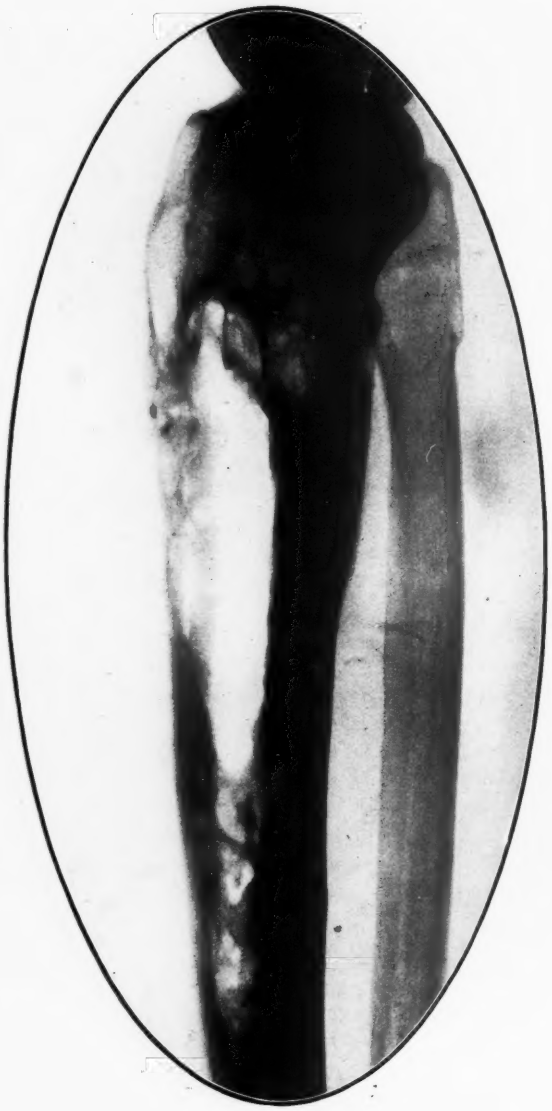

FIGURE XXI.

FIGURE XXII.

ILLUSTRATIONS TO THE ARTICLE BY DR. L. J. CLENDINNEN AND DR. H. F. PRAAGST.

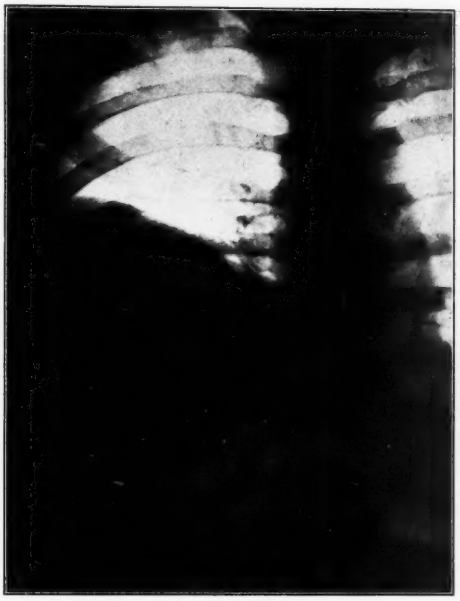

FIGURE I.
Posterior view of chest of patient with sarcoma involving lower third of right lung before treatment.

FIGURE II.
Lateral-posterior-oblique film of patient seen in Figure I, showing the almost circular outline of the mass.

FIGURE III.
Posterior view of chest of same patient as in Figures I and II two months later, following a course of deep X ray therapy.

FIGURE IV.
Posterior view of chest of patient with endothelioma involving apical lobe of left lung.

ILLUSTRATIONS TO THE ARTICLE BY DR. L. J. CLENDINNEN AND DR. H. F. PRAAGST.

FIGURE V.
Posterior view of chest of same patient as in Figure IV six months after first course of deep therapy.

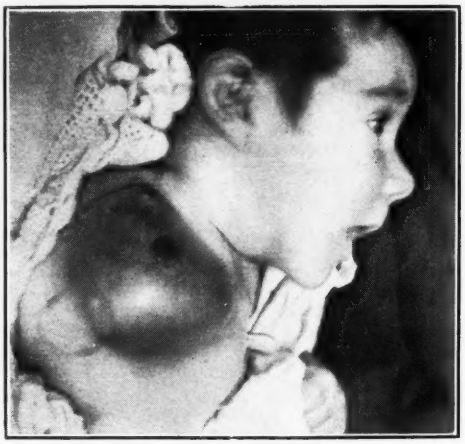

FIGURE VI.
Photograph of baby aged six weeks, with angio-sarcoma of right clavicular region.

FIGURE VII.
Photograph of same patient as in Figure VI at the age of ten and a half months, after treatment.

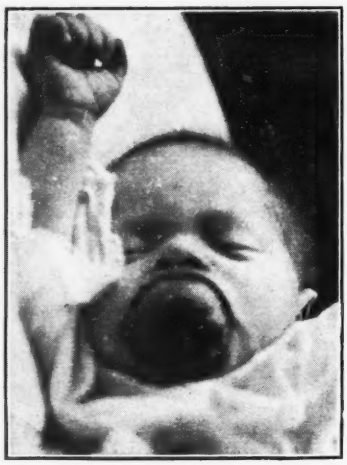

FIGURE VIII.
Photograph of baby aged one week with congenital sarcoma under tongue, two weeks before operation.

FIGURE IX.
Photograph of same patient as in Figure VIII at age of eighteen months, following treatment for recurrence of growth.

ILLUSTRATIONS TO THE ARTICLE BY DR. R. B. WADE AND DR. J. STEIGRAD.

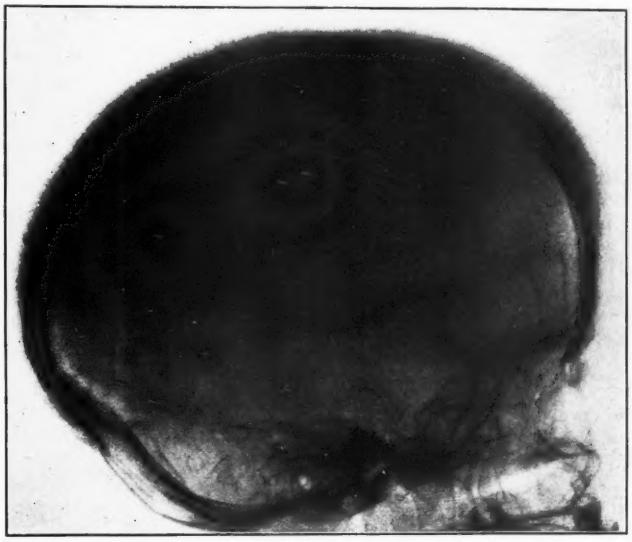

FIGURE I.

FIGURE II.

ILLUSTRATIONS TO THE ARTICLE BY DR. G. RALEIGH WEIGALL AND DR. EDWARD H. DERRICK.

FIGURE I.
Hodgkin's Disease of the Twelfth Thoracic Vertebra and the Eleventh Rib.

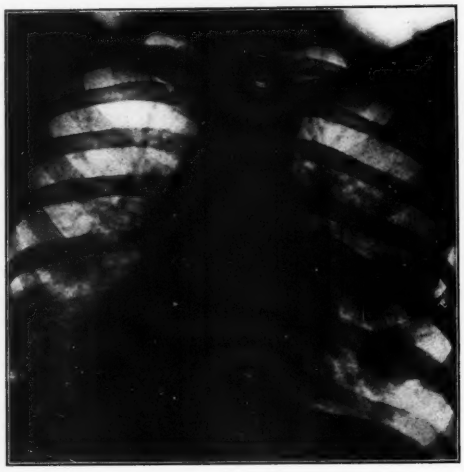

FIGURE II.
Hodgkin's Disease of the Lungs.

ILLUSTRATIONS TO THE ARTICLE BY DR. WOLFE DAVIS AND DR. H. FLECKER.

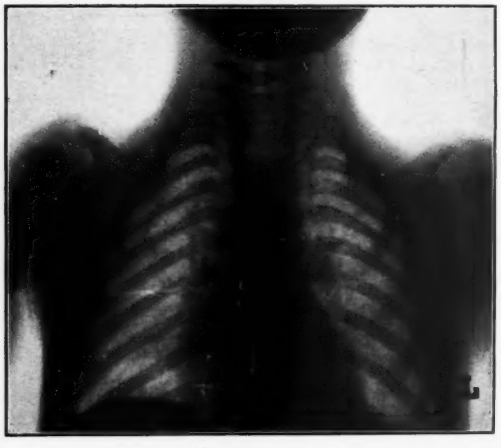

FIGURE I.
Skiagram of Patient's Thorax. No trace of the clavicles can be seen. The free acromium is well shown.

FIGURE II. FIGURE III.

treatment. All my severe infections have occurred after parturition.

There seems to be a definite connexion between careless hygiene and the development of cystitis and pyelitis.

Glycosuria.

Glycosuria means either that the patient is a diabetic or that this condition is only temporary. In border-line cases the blood sugar examination is made two and a half hours after a heavy meal of carbohydrate. If the blood sugar is 160 milligrammes per hundred cubic centimetres, then it is a case of diabetes. If the blood sugar is 90 milligrammes per hundred cubic centimetres, we are dealing with the renal type of glycosuria.

Although the percentage of patients with glycosuria is variously estimated, from 13% to 15% by overseas authorities, in my experience the condition is rare; scarcely 1% of my patients have manifested this reaction. Perhaps, of course, my methods are faulty. I have never had to adopt any treatment beyond dietetic measures and the glycosuria always disappears after the confinement.

This is an interesting subject and I should like to know what is the recognized percentage amongst Australian women. True diabetes I have yet to meet as a complication of pregnancy.

Cardiac Complications.

Cardiac symptoms may be due to mechanical causes and may arise in the later months of pregnancy. The increasing intraabdominal pressure influences the position of the heart, the result being slight dilatation of the right side of the heart. This condition is physiological, but may become pathological in hydramnios, multiple pregnancy or with an abnormally large fœtus when labour must be induced to relieve the cardiac distress.

Cardiac dilatation may be due to toxæmia, usually renal, although I have seen one case of rheumatic fever in a pregnant woman. The treatment forms part of the general treatment of the toxæmia in question.

Patients with valvular lesions, if they are well compensated, require only careful watching and avoidance of excitement, over-exertion and overeating. When symptoms of failing compensation manifest themselves, I put the patient to bed, giving cardiac stimulants and keep her at rest, if possible, until the child is viable. Then I induce labour.

When a patient with any valvular incompetence engages me, I warn her that at any time her pregnancy may have to be terminated. It is most difficult to give a prognosis in these cases. Any signs of failing compensation in the third or fourth month I regard as a contraindication to continuance of the pregnancy.

I have found that the majority of patients with mitral stenosis go through a normal pregnancy, but cardiac distress often developes during labour. In mitral regurgitation the outlook is usually not so good, early induction often being necessary. Aortic insufficiency is the most serious. One patient who refused induction on religious grounds, died four hours after delivery.

In my opinion all these patients require some form of narcosis during the first stage of labour. I use morphine-scapolamine, giving oxygen when required.

No matter how normal a course the pregnancy may run, there is always the possibility of serious cardiac failure during labour. For that reason I never regard any old valvular lesion lightly.

Pelvic Abnormalities.

I reexamine all patients with any degree of contraction either of the brim or outlet at the seventh month to decide how much longer gestation may safely be allowed to continue. I am particularly careful in the case of a funnel-shaped pelvis, as it is so much more difficult to make a satisfactory comparison between the size of the fœtal presenting part and the size of the lower pelvic canal or outlet.

Early induction has given me excellent results in both mother and child. I consider Cæsarean section inexcusable treatment for pelvic contraction, except under exceptional circumstances.

A large pelvis, although offering no obstruction to delivery, ultimately may give rise to greater morbidity than any form of contracted pelvis. I have seen *nulliparæ* with a definite cystocele associated with a large pelvis. Pregnancy exaggerates this condition and the first parturition may result in even the third degree of prolapse.

A generally enlarged pelvis needs careful watching towards the end of pregnancy. The whole uterus often sinks so low that the presenting part lies practically on the perineum during the last fortnight. The patient sometimes complains bitterly of the pressure and discomfort. To relieve her symptoms as well as the strain on the pelvic floor I induce labour two to three weeks before term.

Post Maturity.

Whenever I am satisfied that the period of gestation is complete apart from the possibility of a large fœtus and the strain of waiting is telling on my patient's nerves, I induce labour.

With regard to mal-presentations, except in rare cases of an oblique lie of the fœtus, I leave them alone. A breech is just as easy to deliver as a vertex and a head in the occipito-posterior position will return to that position unless rotated when labour is well established.

Conclusion.

This subject is an enormous one and I have dealt with it most inadequately, but I hope at least you will allow me the virtue of enthusiasm, with, of course, its drawback of misdirected energy.

I probably have the same aims and aspirations as the young English head master who is at present trying to revolutionize education in the great public schools, his slogan being: "Be fervent, boy, be fervent; any fool can be right."

Reports of Cases.

SOME RESULTS OF TREATMENT BY RADIUM AND DEEP X RAY THERAPY.[1]

By L. J. CLENDINNEN, M.B., B.S. (Melbourne),
Honorary Radiologist, Melbourne Hospital, Melbourne,

AND

HOWARD F. PRAAGST, M.B., B.S. (Melbourne),
Honorary Assistant Radiologist, Melbourne Hospital, Melbourne.

THE films taken from two patients with sarcoma involving the lung, having X ray appearances closely resembling those of hydatid cyst, are of interest and illustrate a rapid response to deep X ray therapy.

The first of these two patients, T. McL., a male, aged twenty-six years, was referred from another centre with a history that X ray examination made on account of pain in the chest had revealed what was apparently an hydatid at the base of the right lung. At operation a solid swelling was presenting between the lower ribs which was evidently neoplastic; section showed it to be a round-celled sarcoma. No further surgical procedure was carried out and the patient was referred for radiotherapy. Films taken at this time, April, 1927 (see Figures I and II), showed an almost circular mass, 12·5 centimetres in diameter, with well-defined edge, of density approximating that of an hydatid cyst, occupying the lower half of the right lung in its anterior and lateral portion. There was an area of bone absorption and irregular proliferation in the anterior axillary portion of the fifth rib, six centimetres in length, with a second area of absorption without proliferation in the sixth rib. The primary condition was evidently an osteogenic sarcoma of the fifth rib with involvement of lung and sixth rib. A course of deep therapy was given during the ensuing four weeks and films taken in July, 1927, a month after the last treatment, revealed definite reduction in the size of the mass (see Figure III) coincident with great improvement in the patient's clinical condition.

The second patient, H.W.T., a female, aged forty-five years, was referred on October 6, 1927, for X ray examination of the chest. There was a rounded mass, nine centimetres in diameter (see Figure IV), with a well-defined border, occupying the posterior two-thirds of the left apical lobe, extending mesially almost to the mid-line and downwards as far as the seventh rib posteriorly. The density of the mass was slightly greater than that of the usual hydatid cyst and the possibility of neoplasm was considered, but the fact that the patient lived in an area notoriously hydatid infested, influenced the diagnosis in favour of hydatid. On further investigation the Casoni and complement fixation tests for hydatid and the Wassermann test yielded no reactions. At operation the mass was found to be solid with a broad pedicle attaching it to the transverse processes of the vertebræ. A section was taken which proved to be "endothelioma, probably of the pleura." Deep therapy was given anteriorly and posteriorly on November 21 and 25 and films taken six weeks later revealed a striking diminution in the size and density of the growth. Further therapy was given later and her most recent film, May, 1928 (see Figure V), showed that the diminution has been maintained.

Baby H.C., male, aged six weeks, was referred from the Children's Hospital in January, 1927, with a very soft, rapidly growing, non-fluctuant mass about the size of a coconut in the right clavicular region, causing pressure on and displacing the trachea and œsophagus (see Figure VI). It was clinically an angiosarcoma. Deep therapy was given which caused considerable reduction of the mass, but after two months further activity was shown and radium needles were buried into the growth which was still exceedingly vascular. Further X ray treatment was given in April, after which the growth disappeared. This patient was shown at the meeting of the Victorian Branch of the British Medical Association at the Melbourne Hospital on May 18, 1927, and remains well at present, having had no treatment for over thirteen months. As his home is three hundred miles from Melbourne, a photograph taken at the age of ten and a half months is reproduced (see Figure VII).

Baby M.C., female, aged six weeks, was referred for radiotherapy for recurrent sarcoma under the tongue. There was a history of a congenital growth under the tongue (see Figure VIII) which rapidly increased in size and was removed when the child was three weeks of age. Section showed it to be rhabdo-myo-sarcoma. Three weeks later the growth had recurred to the size of a walnut. Deep therapy and radium treatment were given over a period of four months, when the growth disappeared. A recent photograph of the child at eighteen months (Figure IX) is reproduced. The mother states in a letter that apart from slight limitation of movement, the tongue appears normal.

[1] The patients described herein were shown at a meeting of the Victorian Branch of the British Medical Association on May 16, 1928.

SPLENOMEGALIC JAUNDICE WITH ASSOCIATED CHANGES IN THE SKULL.

By R. B. WADE, M.D., Ch.M. (Sydney), F.C.S.A.,
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AND

J. STEIGRAD, M.B., Ch.M. (Sydney),
Resident Medical Officer, Royal Alexandra Hospital for Children, Sydney.

DICK C., aged six years, and Fred C., aged five years, were admitted to hospital. The only symptoms complained of were those of tiring easily and of always being pale and sallow. Both had had attacks of jaundice, diagnosed as catarrhal jaundice, a few months before admission.

There are four children in the family. Three boys all suffer with splenomegaly and jaundice. (One of these had died of an intercurrent pneumonia.) One girl is quite healthy. No illness has occurred in the parents or their relatives.

On examination both appeared definitely anæmic with an icteric tinge. They both had advanced dental caries, but no Hutchinson's teeth. Both showed a peculiar condition of the skull, namely, frontal and parietal bosses with furrows in between giving a "hot cross bun" effect. Their spleens were palpable, that of Dick enlarged nearly to the umbilicus and Fred's just palpable in the left hypochondrium. The liver margin in the elder boy was palpable five centimetres (two inches) below the costal margin; Fred's liver was not palpable at all. There was no ascites in either boy nor were any glands palpable in their necks, groins or axillæ. During their ten days in hospital Dick's temperature at some part of the day would reach 37·8° C. (100° F.). Fred's temperature remained normal except on three occasions, when it rose to a maximum between 38·3° and 38·9° C. (101° and 102° F.).

The following pathological examinations were carried out:

The Wassermann test in both instances yielded negative reaction.

The Van den Bergh test in each yielded a delayed direct reaction.

The Bence-Jones albumose test failed to demonstrate any abnormal protein and chemical and microscopical examination of the urine revealed no abnormality.

The blood counts on November 6, 1927, were as follows:

Dick:	Red cells, per cubic millimetre ..	2,000,000
	Hæmoglobin value	40%
	Colour index	10
	Corpuscle index	40

There was much diffuse polychromasia, anisocytosis and poikilocytosis were present and four megaloblasts were seen.

The leucocytes numbered 8,500 per cubic millimetre. Polymorphonuclear cells numbered 68% or 5,780 per cubic millimetre; lymphocytes, 25% or 2,125 per cubic millimetre; monocytes, 50% or 425 per cubic millimetre; eosinophile cells, 2% or 170 per cubic millimetre.

The blood platelets were 280,000 per cubic millimetre.

Fred:	Red cells, per cubic millimetre ..	1,800,000
	Hæmoglobin value	45%
	Colour index	1·2
	Corpuscle index	36

Diffuse polychromasia, anisocytosis and poikilocytosis were present.

The leucocytes numbered 7,000 per cubic millimetre. Polymorphonuclear cells numbered 58% or 4,060 per cubic millimetre; lymphocytes, 38% or 2,660 per cubic millimetre; monocytes, 4% or 280 per cubic millimetre.

No eosinophile cells were seen in a count of 200 leucocytes. Blood platelets were 144,000 per cubic millimetre.

The honorary radiographer reported concerning the X ray appearance of the skulls and long bones. The latter, namely, the femur, humerus, tibia and fibula in each case showed no obvious abnormality. The skulls, however, manifested a diffuse overgrowth of the frontals and parietals, reported as probably the diffuse osteitic type of *leontiasis ossea.*

On March 20, 1928, the brothers were readmitted to hospital. Their condition was much the same as before, but both appeared slightly more anæmic than four months previously. Dick's liver and spleen were slightly smaller, but still noticeably enlarged and Fred's liver had become palpable.

On this occasion the Van den Bergh tests yielded a negative immediate direct reaction and a positive indirect reaction. The coagulation times for their blood were: Dick one and three quarter minutes in a capillary tube at room temperature, and Fred, two minutes. The fragility tests gave normal results. The blood counts at this time, March 28, 1928, were as follows:

Dick:	Red cells, per cubic millimetre ..	1,400,000
	Hæmoglobin value	30%
	Colour index	1·03
	Corpuscle index	28

Diffuse polychromasia and much anisocytosis in the form of macrocytes were seen, but the shape of the cells on this occasion was fairly regular. No megaloblasts or normoblasts were seen.

The white cells numbered 6,400 per cubic millimetre. Polymorphonuclear cells numbered 56% or 3,524 per cubic millimetre; lymphocytes, 35% or 2,240 per cubic millimetre; monocytes, 7% or 448 per cubic millimetre; eosinophile cells, 2% or 120 per cubic millimetre; blood platelets, 70,000 per cubic millimetre.

Fred:	Red corpuscles, per cubic millimetre	1,680,000
	Hæmoglobin value	35%
	Colour index	1·06
	Corpuscle index	33

As before, diffuse polychrome staining and anisocytosis (macrocytes) were present, but no definite poikilocytosis. There were no abnormal red cells.

The white cells were 6,200 per cubic millimetre. Polymorphonuclear cells numbered 51% or 3,162 per cubic millimetre; lymphocytes, 36% or 2,432 per cubic millimetre; monocytes, 10% or 420 per cubic millimetre; eosinophile cells, 3% or 186 per cubic millimetre; platelets, 58,800 per cubic millimetre.

The honorary radiographer reported that the skulls were slightly thicker than four months previously and that there was now some enlargement of the trabeculæ in the shafts of some of the long bones.

Discussion.

Acholuric hæmolytic jaundice or familial splenomegaly, as the name implies, attacks several members of the same family. In this case the boys were attacked and the girl was missed. The spleens in the two cases were not enormous and were variable in size. Jaundice of the hæmatogenous type was present. No increased fragility of the red corpuscles was found, though this is supposed to be one of the striking points of the condition.

Anæmia was pronounced, but there was no great increase in white cells nor did the blood examination reveal any definite changes of a primary anæmia. The change in certain bones of the skull was one of the most striking features.

Cooley, Witwer and Lee[1] describe seven cases of familial splenomegaly in which a very similar bone condition was present. In their cases the blood condition was similar to that of a von Jaksch's anæmia.

The Radiological Aspect.

We are indebted to Dr. H. R. Sear for the following commentary from the radiological point of view.

> The three cardinal features of the disease under discussion appear to be anæmia, enlargement of the spleen and changes in the osseous system.
>
> It is the type of change in the osseous system, however, that serves to differentiate this lesion from other conditions that may show bony changes, anæmia and splenic enlargement. For example, as Thomson has shown, advanced rickets and again Albers Schönberg's disease both may affect these three systems, but the bone changes are quite distinct. In Albers Schönberg's disease it is the long bones essentially that are involved, whereas here the main change is in the squamous bones of the cranial vault.
>
> Before proceeding to a more minute examination of the bones, I might mention that these two children show such similar abnormalities that a description of one would serve for the other.
>
> The bones showing the most pronounced involvement are the frontals and parietals, the occipital being free from gross pathological change in both cases. The floor of the anterior cranial fossa appears to be very slightly thickened, the facial bones show no change.
>
> The parietals and frontals are greatly thickened, the degree of thickening being most pronounced in the central parts of the bones and gradually decreasing as the sutures are approached. The fronto-parietal sutures have in both children undergone synostosis and the parieto-occipital sutures are partially obliterated.
>
> The bones show no definite differentiation in the outer and inner tables and diploe, though there is a suggestion of a very thin inner table. No outer table can be seen and the bones display radiographically a most peculiar vertical striation, more pronounced as we approach the outer surface, the whole appearance suggesting that the osseous tissue would be soft and vascular, as appears to be the case in the few patients that have come to *post mortem* examination. These features are seen in the accompanying illustrations (see Figures I and II).
>
> It is this vertical striation that differentiates it from all other lesions; it is remarkably even and uniform. When these cases first came under review, the possibility was raised as to whether the condition belonged to one of the rare subdivisions of *leontiasis ossea*, namely, the diffuse osteitic type, such as the Bristol skull or the Jadelot or Sacy skull. This lesion shows great thickening of the cranial bones (*osteitis fibrosa*) with obliteration of the cranial sutures; but with enlargement of the mandible and facial bones; it was thought that possibly they might be early stages of such a condition. Again there is a condition described by some Continental writers as diffuse hyperostosis of the cranial bones, under which this condition might be grouped radiographically; but the other clinical features are such that it would appear to be a definite clinical entity.
>
> The long bones in this lesion are supposed to show certain changes; they are said to be more transradiant and the Haversian canals wider; the long bones in these two children, however, showed very slight changes, possibly a slightly looser reticulation of the

Haversian system, especially in the ends of the long bones and in the metacarpals and metatarsals; but the changes in these two children were extremely slight and not nearly so pronounced as may be seen in some of the diseases of the spleen and blood, such as Gaucher's disease or some of the leuchæmias.

Reference.

(1) T. B. Cooley, E. R. Witwer, P. Lee: "Anemia in Children with Splenomegaly and Peculiar Changes in Bones; Report of Cases," *American Journal of Diseases of Children*, September, 1927, page 347.

A CASE OF HODGKIN'S DISEASE WITH INVASION OF BONE, LUNG AND OTHER ORGANS, SIMULATING TUBERCULOSIS.[1]

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THE following case appears worthy of recording in some detail on account of the rarity of reported cases of a similar nature and as an example of a condition the diagnosis of which at first appeared fairly obvious, but was later proved completely wrong.

History and Course of Illness.

Mrs. C.B.C., aged thirty-one, was admitted to hospital on October 5, 1927, with the following history: When aged fifteen, she suffered from cough, hæmoptysis and loss of weight. She came of a family with a strong tuberculous history and was treated for a year in a sanatorium in England. Tubercle bacilli were found in her sputum. She improved considerably, was regarded as cured and completed the training course of a nurse at a hospital in England without further illness. She married and came to this country three years ago.

Two and a half years before admission, just before the birth of her only child, she noticed several lumps in her neck, which were diagnosed as tuberculous glands. She had X ray therapy and they decreased in size and became more adherent. A year before admission she first complained of pain in the back and pains in the abdomen which occasionally passed down the right leg. X ray examination revealed caries of the twelfth thoracic vertebra. A cough appeared, she lost weight and had night sweats. About the same time the glands of her neck enlarged. She was then transferred to this hospital with the provisional diagnosis of tuberculous lymphadenitis and tuberculous disease of the spine.

On admission she was pale and wasted, running a regular evening temperature of 37·8° to 38·3° C. (100° to 101° F.). Her lungs appeared normal, except for one patch of relative dulness in the right axilla, where the breath sounds were slightly diminished and raised in pitch. A skiagram of her chest at this time was reported as showing "limited fibrotic changes of right middle lobe; slight enlargement of glands at left hilum; several discrete nodules (healed tuberculous glands) about left hilum."

There was a definite kyphosis of her spine in the lower thoracic region, the spinous processes of the eleventh and twelfth thoracic vertebræ being the most prominent. There was limitation of movement of this segment of the spine and some tenderness. She complained of pain of varying intensity, passing round the abdomen in the region of the umbilicus and passing down the left groin and thigh. No abnormal neurological signs were detected. An X ray examination of her spine at this time revealed caries of the twelfth thoracic vertebra with partial collapse. The vertebral extremity of the eleventh right rib was also involved. It showed considerable enlargement with large areas of absorption and a few spicules of new bone. Although the radiogram of the vertebra seemed to confirm the diagnosis of tuberculosis, the appearance of the rib suggested rather malignant disease.

The illustration (Figure I) was made from the excised *post mortem* specimen and shows the appearance after the disease had progressed for a further four months.

On both sides of her neck were matted masses of firmly adherent lymphatic glands. She said that they had become much smaller and much less tender since the X ray therapy some months before. There were on admission no other demonstrable glands. Her liver was not enlarged, nor her spleen palpable. Blood examination revealed only a secondary anæmia of moderate severity. Her blood serum failed to react to the Wassermann test.

Her spine was immobilized in a plaster bed with relief of the root pains and a temporary improvement in her general condition.

About a month later a single discrete gland became palpable in her left axilla and enlarged rapidly. On November 20 it was about as large as an almond and was excised under local anæsthesia. Microscopical examination of the gland revealed a characteristic picture of Hodgkin's disease. The lymphoid tissue had almost entirely disappeared, there was proliferation of the reticular cells with numerous Dorothy Reed giant cells, areas of commencing fibrosis and infiltration with eosinophile and neutrophile leucocytes.

Other groups of lymphatic glands became enlarged in a short time, being palpable in both axillæ and groins, and through her wasted abdominal wall masses of retroperitoneal glands could be felt. The liver enlarged and the spleen became easily palpable.

She was running a regular temperature of 37·8° to 38·9° C. (100° to 102° F.) and rapidly became cachectic. Vomiting was a troublesome symptom and could be relieved by nothing but large doses of morphine. During December she developed a dyspnœa and this and the cough became steadily worse. Examination of her lungs revealed the physical signs of patchy consolidation on both sides. The skiagram (December 14, 1927) shows the condition of the chest at this time (see Figure II).

The above symptoms and her anæmia steadily progressed and she died in a very cachectic condition on February 12, 1928.

Post Mortem Notes.

At the *post mortem* examination the cervical glands were found to be numerous, firm and slightly enlarged.

The lower parts of both lungs were densely adherent to the parieties and diaphragm. When the lungs were removed, the adhesion was seen to be due to a sheet of firm white infiltration covering the internal surfaces of the last six ribs and intercostal muscles. The left lower lobe and the two lowest lobes on the right were almost solid. On section the cut surface appeared mostly grey and firm with yellowish areas of breaking down. The upper lobe of each lung was unaffected except for an old fibrous scar near the left apex, which confirmed the story of an earlier tuberculous infection. The bronchial and mediastinal glands were infiltrated and enlarged.

The glands around the pancreas were enlarged, those along the abdominal aorta greatly enlarged, especially on the left side, and were matted together by diffuse infiltration into a mass which surrounded the left suprarenal gland and extended into the hilum of the left kidney. These glands on section were mostly white and firm with small yellow caseous areas. The spleen weighed 283·5 grammes (ten ounces) and was the characteristic "hard-bake" spleen with rosette-like aggregations of small white nodules.

The vertebral column from the eighth thoracic to the fourth lumbar vertebra was removed in a mass, examined by X rays and then sawn sagittally. The body of the twelfth thoracic vertebra was seen to be compressed, causing a kyphosis as well as a lateral angularity. The normal red *stratum spongiosum* of the body of this vertebra was replaced partly by dense bone and partly by soft grey and yellow tissue. The intervertebral disc below was

[1] The patient described herein was seen by the authors when they were respectively resident medical officer and resident pathologist at the Austin Hospital, Melbourne.

intact, that above partly infiltrated with the soft tissue which had also begun to invade the lower part of the eleventh vertebra. The spinal canal was slightly narrowed, but the cord and the internal surface of the *dura mater* were unaffected. There was a round, soft enlargement of the vertebral end of the eleventh right rib.

The heart, liver, kidneys, right suprarenal gland, pancreas, stomach and intestines were not apparently involved.

Microscopical examination was undertaken of sections, among others, from lung, spleen, bronchial gland, aortic gland, left suprarenal, twelfth thoracic vertebra and the rib swelling. In all of these the appearance was of Hodgkin's disease and similar to that of the excised axillary gland, except that the process in many places was more advanced, with wide tracts of fibrosis and large areas of necrosis. Sections of lung, aortic gland, rib and vertebra were searched for tubercle bacilli without success.

Discussion.

The features of this case are as follows:

1. The partial destruction of a vertebra with collapse. Invasion of bone marrow is a frequent finding in Hodgkin's disease. Turnbull[1] observed it in nineteen cases out of thirty-nine and Symmers[2] in seven cases out of fourteen. In our case not only was the bone marrow invaded, but the osseous tissue also and the structure of the vertebra so interfered with as to cause partial collapse. This is less common. Ewing[3] states that while bone marrow lesions are commonly observed, as a rule the bone tissue and periosteum remain intact. Several authors have observed bony changes—Arnell,[4] Hultén,[5] Dresser[6] and Louste[7] by radiographical and Cone[8] by microscopical examination. We have not, however, found any previous record of a case in which collapse of a vertebra became a leading clinical feature.

2. The massive infiltration of the lungs.

3. The diagnostic trap. At the time of admission of this patient the angular kyphosis, limitation of movement, pains in the back and tenderness pointed strongly to tuberculous spondylitis. The cough, loss of weight, night sweats, evening temperature and the early chest signs seemed undoubtedly tuberculous. The combination of both the spine and chest conditions in a young woman with a previous tuberculous infection made a diagnosis of tuberculosis irresistible. Yet it was wrong. First, the radiogram questioned the tuberculous nature of the rib swelling, later the histology of the excised gland revealed the presence of Hodgkin's disease and finally the autopsy proved the whole process to be Hodgkin's disease and failed to disclose any sign of recent tuberculosis.

Acknowledgement.

We desire to thank Mr. H. C. Trumble, Honorary Surgeon, for permission to publish this case, also Dr. R. A. Willis, Medical Superintendent, for his assistance.

References.

(1) H. M. Turnbull, quoted by H. Rolleston: "Lymphadenoma," *The Lancet*, December 12, 1925, page 1209.

(2) D. Symmers: "The Clinical Significance of the Pathological Changes in Hodgkin's Disease," *American Journal of the Medical Sciences*, February, 1924, pages 157 and 313.

(3) J. Ewing: "Neoplastic Diseases," 1922, page 371.

(4) S. Arnell: "Hodgkin's Disease with Bone Destruction —Two Cases," *Acta Radiologica*, June, 1927, page 259.

(5) O. Hultén: " ," *Acta Radiologica*, June, 1927, page 245.

(6) R. Dresser: "Lymphoblastoma (Hodgkin's Disease) of the Sternum," *American Journal of Roentgenology and Radium Therapy*, June, 1926, page 525.

(7) Louste, Thibaut and Valière-Vialeix: "On a Case of Hodgkin's Disease Which Presented During its Course Osseous Lesions (Vertebral and Sternal) of Difficult Interpretation," abstracted in the *American Journal of Roentgenology and Radium Therapy*, February, 1925, page 192.

(8) S. M. Cone: "Bone in Hodgkin's Disease," *Journal of Bone and Joint Surgery*, July, 1927, page 458.

CONGENITAL ABSENCE OF THE CLAVICLES (HEREDITARY CLEIDOCRANIAL DYSTOSIS).

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L.G., a male, aged five years, was admitted to the Homœopathic Hospital for the purpose of circumcision and in the course of routine examination a most interesting set of abnormalities was discovered, peculiar to a condition named by French writers *dystose cleido-cranienne héréditaire*.

Under this heading four main features of the condition are described: (i) Clavicular, (ii) cranial, (iii) dental, (iv) hereditary. Three of these are well exemplified in this case.

The clavicles were first thought to be totally absent, as no trace of them could be seen in the X ray photograph (see Figure I), but further palpation revealed the presence of small bilateral cartilaginous stubs 1·25 centimetres (half an inch) in length attached to the sternum.

The acromion processes of both scapulæ were rudimentary and ill-defined, allowing the head of the humerus to be palpated from above. The sterno-cleido-mastoid muscle had its origin from the first rib, while the *pectoralis major*, *trapezius* and deltoid muscles had no clavicular attachments.

The upper limbs at the shoulder joints had an extraordinary wide range of movements and the shoulders could be brought forward so that the deltoid eminences touched (see Figure III). There was no deficiency of power nor any disability caused by the missing clavicles, the child being able to climb fences and trees and play with boys of his own age.

Cranial defects similar to those described by other writers were present. The anterior fontanelle was widely patent and the suture lines ununited, the frontal suture lines being eight millimetres (one-third of an inch) wide, while the circumference of the skull was fifty-two centimetres (twenty and three-quarter inches).

According to Pillsbury dental abnormalities are common, both in the primary and secondary dentitions, the primary teeth persisting until twelve years of age. In this case, however, the primary teeth were already lost owing to caries.

The hereditary nature of the condition was emphasized by a similar abnormality of the skull (but not of the clavicles) existing in the grandfather, although the parents, brothers and sisters are reported to be perfectly developed and to have no trace of any abnormality.

The child was born at full time and the mother had a particularly easy labour, no instruments being used. The boy has always been very healthy and is very bright and mentally alert and of normal physique (see Figure II).

The condition is interesting inasmuch as it is so rare, only sixty cases having been reported in England, France, Denmark, Germany and the United States of America since Strahmann in 1857 first described the condition.

In most cases the abnormality has only been discovered by the medical practitioner in the course of routine examination.

Reviews.

CLINICAL BACTERIOLOGY.

"Systemic Infections," by Dr. A. Knyvett Gordon, is a plea for the more effective cooperation of the bacteriologist with the clinician in the treatment of infections, especially chronic infections.[1]

Unfortunately in so small a compass it is difficult to give definite proofs for the statements made and it is doubtful whether the work would carry conviction to anyone who was not already convinced of the soundness of the views expressed. However, it may have some value to those who want help in the methods employed in studying cases of chronic infection.

The chief value of the book must be in stimulating an interest in such conditions; those who require such stimulation, are more likely to turn to magazine articles than to books. The bibliography is small and not in any way comprehensive.

THE ELEMENTS OF PHYSIOLOGY.

"A Synopsis of Physiology," by A. Rendle Short and C. I. Ham, is distinctly above the usual average of synopses.[2] It is always very much better for a student to prepare his own synopsis of any subject, for the mere process of extracting the important essentials makes him read his text book carefully and the sentences he uses in the synopsis, will call to mind details which he has not included. For those who for some reason or other prefer a synopsis prepared by someone else this book should be very useful for it is sound, modern and moderately comprehensive.

In the preface it is stated that "practitioners who wish to keep abreast of modern physiology will find here a means of making a quick survey." It is doubtful whether they would benefit to the extent the authors consider, for while the information conveyed is succinct and on the whole accurate, it is rather too much condensed and the book used alone might easily lead astray. For example, "Starling's law—power of contraction varies with the stretch of the fibres during diastole," by itself means very little and might easily be misinterpreted.

As a text book of physiology it is poor, even for a student who "attends a good course of lectures," but as a synopsis it is on the whole good.

A LABORATORY HAND BOOK.

E. R. Stitt's well known hand book attempts within the compass of a single volume of very moderate size to be a staff and guide in bacteriology, pathology and all the branches of parasitology.[3] A remarkable measure of success is achieved in this extremely difficult task. The book is a mass of well chosen and well packed information and it is among the best of its kind in any language. Those who have had practical experience of the work of a general laboratory, especially of an isolated laboratory, whether tropical or not, can testify to the constant usefulness of the book.

The eighth edition is slightly larger than the previous one and consists of about 850 pages, much of which is in small print. The revision has been extensive and a large amount of new matter appears.

Many specialists, most of them American, have collaborated in the production of the work.

Great prominence is given to the Kahn test and a full account of the method and its varieties prepared by Dr. Kahn is included. Valuable space is even devoted to photographs of laboratory workers performing the test. Stitt considers this test to be now the most suitable method in the serological diagnosis of syphilis.

In the bacteriological section both the new nomenclature of the Society of American Bacteriologists and the old familiar names are given.

The sections on scarlet fever, on apparatus for counting blood corpuscles, on the sedimentation test, on sickle-cell anæmia and on tests of liver function, are a few only of those dealing with recent advances in knowledge and technique.

In the parasitological sections there are many changes in zoological nomenclature. This is a necessary evil and older or better known names are given as well as those believed to be zoologically correct at the time the work was prepared. Some of the latter of course already require revision. At the present time it is often easier to discover what a parasite is than what is should be called. The numerous illustrations are usually useful and well chosen, being designed to help the medical man and not for the zoological specialist.

A perusal of these parasitological sections suggests some minor criticisms; but the extreme compression is doubtless often responsible. The paragraphs on the *Trichostrongylidæ*, for instance, are not satisfactory and it seems a pity to classify the organism of rat-bite fever with the *Leptospiræ*, even though the text indicates that there is no resemblance between them.

The same subject is often dealt with in more than one part of the book and the index is not invariably an adequate guide.

On the whole the book has gained greatly in usefulness by the revision and will retain its place among the well-worn objects lying on the laboratory bench.

SECTION CUTTING.

Miss Frances Ballantyne, in "An Introduction to the Technique of Section Cutting" has at the suggestion of Professor J. Graham Kerr arranged in book form the notes of the late Peter Jamieson on the methods employed in the Department of Zoology in the University of Glasgow.[1] The work is a small one, the price moderate and the scope covers the technique of paraffin and celloidin imbedding with the necessary fixation and appropriate staining, whether in bulk or of individual sections, singly or in series. The notes are intended more particularly for workers in zoology including embryology and consequently do not deal with frozen sections, often employed by pathologists for the purpose of rapid diagnosis, or the special staining methods for the nervous system and other tissues required for medical purposes. Nevertheless the medical practitioner will find within its pages useful hints and helpful suggestions, the outcome of an extensive experience by a practised microscopist, that will save him time and trouble and improve his technique and consequently his results.

[1] "Systematic Infections: Their Diagnosis and Treatment," by A. Knyvett Gordon, M.B., B.C., B.A. (Cantab.); 1928. London: Baillière, Tindall and Cox. Demy 8vo., pp. 186. Price: 10s. 6d. net.

[2] "A Synopsis of Physiology," by A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S., and C. I. Ham, M.B., B.Ch., M.R.C.S., L.R.C.P.; 1927. Bristol: John Wright and Sons, Limited. Crown 8vo., pp. 258, with illustrations. Price: 10s. 6d. net.

[3] "Practical Bacteriology, Blood Work and Animal Parasitology, including Bacteriological Keys, Zoological Tables and Explanatory Clinical Notes: A Compendium for Internists," by E. R. Stitt, A.B., Ph.G., M.D., Sc.D., LL.D.; Eighth Edition, Revised and Enlarged; 1927. Philadelphia: P. Blakiston's Son and Company. Post 8vo., pp. 852 with illustrations. Price: $6.00 net.

[1] "An Introduction to the Technique of Section-Cutting," from the notes of the late Mr. Peter Jamieson; Edited by Frances M. Ballantyne, M.A.; 1928. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 80. Price: 3s. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 27, 1928.

The Bosch Bequest.

The Medical School of the University of Sydney is about to enter upon a new period of its existence, thanks to the munificence of Mr. George H. Bosch, a wealthy citizen of Sydney. For some time Mr. Bosch has been closely associated with the work in the Medical School and has shown his live interest by the endowment of medical research. Last year he gave the sum of £27,000 to the University for the purpose of founding a chair of embryology and histology and for the promotion of medical research. He has followed the investigations of Dr. N. D. Royle with attention and has manifested more understanding of physiological and pathological problems than is usually evinced by persons who have not studied medicine. On October 11, 1928, the announcement was made at a dinner to celebrate the seventy-sixth anniversary of the foundation of the University of Sydney that Mr. Bosch had given to the University property valued at £220,000 for the specific purpose of establishing three chairs in the Medical School and of equipping the necessary laboratories. Hitherto the chairs of medicine and surgery have been filled by part-time professors. The new professors of medicine, surgery and bacteriology will be required to devote their whole time to their onerous tasks. It is further stipulated that the professors shall engage in research. We understand that the salaries of the new professors of medicine and surgery will be £3,000 a year, in order that the positions may be attractive to men of eminence and exceptional ability. In making the announcement Sir William Cullen, the Chancellor of the University, spoke in terms of the fullest recognition of the magnificent generosity and munificence of the donor. We echo these expressions of gratitude. Mr. Bosch's great donation means much more than a mere gift to a university. It ranks with those rare endowments like that of Lord Iveagh to the Lister Institute of Preventive Medicine, in connexion with which the donor has not only determined the specific purpose of his gift, but has also taken an active part in the management of an institution. He has been wise in selecting medical education and research as worthy objects of support and in endowing them at a time when he has right to anticipate that he will be able to appreciate the fruits of his beneficence for many years. In 1919 the University of Sydney, the University of Queensland and various hospitals benefited under the testament of the late Samuel McCaughey. The University of Sydney received £465,000. We venture to express the opinion that Mr. Bosch is doing more for humanity and for the University of Sydney than did the late Samuel McCaughey, not only because of the specific conditions of his gift, but chiefly because he will be able to measure the usefulness of his donation and to bring his influence to bear on the administration of the fund in the light of experience.

The three medical schools in Australia suffer from the results of a defective system. This system, no doubt, has been adopted in order that the best use may be made of restricted funds. In the past the defect of the system was minimized by the fact that the schools were being developed and controlled by men of extraordinary ability and organizing power. Anderson Stuart, Harry Allen and Edward Stirling have passed hence and the schools that had become famous under their wise direction, have grown out of recognition in size and complexity. It is no longer possible to maintain a modern medical school with one dean and three or four whole-time professors. Today the medical school should be organized and managed by a dean, but the several departments should be placed in the charge of whole-time organizers of proven ability, each of whom should be responsible for the acquisition and dissemination of the knowledge appertaining to his department. We have urged for many years that there should be chairs of the fundamental sciences of bio-chemistry and bio-physics and biology or anatomy and chairs of the ultimate sciences of medicine, surgery and preventive medicine. In addition, subsidiary chairs of obstetrics, bacteriology and pathology are essential. In the next place it is necessary to recognize that each of the professors of the fundamental sciences and each of the

professors of the ultimate sciences should be charged with the organization and control of the teaching of their special subjects by competent lecturers and demonstrators. It is impossible for one man to coordinate the work of all his lieutenants, to control their methods, to determine what new work is to be infused into current teaching, to conduct original research and to organize and lead research investigations by others and at the same time to be the chief lecturer of his department. No greater mistake can be made than to appoint a competent and learned man to be a professor and to compel him to convert his office into that of a glorified lecturer. The Professor of Medicine should be the inspiring head, the director of the team of lecturers and teachers, the authority in charge, but he should refrain from delivering courses of lectures, although it might even be advantageous if he addressed the students and the teaching staff at the beginning of a session or on some special occasion. In the research laboratory or in the clinic he should be freed from all routine work and he should supervise, direct and inspire rather than carry out manipulations and give instruction that can be carried out and given equally well by one of his staff. He should, of course, expound the results of his own researches and use his authoritative position to establish truths that he may have discovered. The same applies to all the other professors, both of the fundamental sciences and of the ultimate sciences. This new system has been rendered feasible in the Medical School of the University of Sydney by the gift of Mr. G. H. Bosch. It is in consequence no exaggeration to state that no one has done a greater service to this University in regard to medical education and the accumulation of medical knowledge than he.

Current Comment.

DRAINAGE OF THE CENTRAL NERVOUS SYSTEM.

The use of intravenous injections of hypertonic salt solution in the treatment of various conditions affecting the central nervous system has been discussed in these pages on several occasions. It is held that the beneficial results are due to a change in volume of the brain consequent on the removal of fluid by osmosis from the tissues to the blood stream. It will be remembered that it has been claimed that this method of treatment has been successful in the treatment of persons suffering from acute poliomyelitis. T. C. Smith claimed that the increase in pressure caused by œdema of the affected cord areas is in a large measure responsible for the paralyses which occur in the early stage of the disease and which disappear later on (see The Medical Journal of Australia, May 8, 1926). He thought that if the pressure could be diminished at the critical moment of the disease, the acute stage might be passed and the patient enabled to recover. It is obvious that in these circumstances a great deal will depend on the state of the kidneys, for unless a great deal of the excess of fluid in the blood plasma were eliminated, the tissues of the body, including those of the central nervous system, would soon return to their former state.

If water be given in excess or if hypotonic saline solution be injected intravenously, the tissues will take up fluid and become œdematous. L. S. Kubie has shown that if at the same time the cerebro-spinal fluid is allowed to escape, the formation of cerebro-spinal fluid is accelerated. He describes this as forced drainage. He also found that if "forced drainage" was applied to an animal suffering from encephalo-myelitis, there occurred an extrusion of the perivascular cellular exudates from the depths of the tissue into the subarachnoid space. He has discussed this subject again.[1]

He states that the possibility of instituting in this way a physiological lavage of the central nervous system with a partial washing out of inflammatory products from the depths of the tissues to their surfaces, "arouses a hope of therapeutic application." He is disturbed, however, at the possibility that the procedure of forced drainage may result in a dangerous increase of intracranial pressure. He has made further observations for the purpose of showing that during forced drainage there is an entirely negligible rise in intracranial pressure and that no demonstrable hydration of the tissues of the central nervous system results. Before describing his recent experiments he points out that observations carried out by himself on a series of normal dogs, have shown that during forced drainage the intracranial pressure did not rise above and only rarely reached the initial normal level. He also adds that he has made observations, shortly to be published, on patients who were suffering from various forms of infection of the central nervous system. On these no direct measurements of intracranial pressure could be made, if the cerebro-spinal fluid was to be allowed to escape freely. He assumed, however, that any significant rise of intracranial pressure would be due to a trapping of fluid in the ventricles and to a diffuse hydration of tissue. In these circumstances compression of the jugular veins would give the characteristic effect of spinal block with little or no output of fluid and little or no rise in pressure in a manometer attached to the lumbar needle. In no instance was there any evidence of the existence of "block." Kubie states that while these findings are

[1] *Brain*, June, 1928.

reassuring, they are not perhaps conclusive. In the article under review, he offers observations on the absence of hydration of the nervous system during forced drainage.

His experiments were made on eight normal rabbits and four dogs under sodium-veronal anæsthesia. The animals were taken in pairs and were given identical injections of hypotonic saline solution. The strength of the solution varied from 0·35% to 0·45% and the amount from 250 to 750 cubic centimetres, according to the weight of the animal. In one of each pair of animals the cerebro-spinal fluid was allowed to flow freely from the *cisterna magna* and in the other no drainage was established. It was found that the venous pressure was not raised materially if the injections were made slowly. When no drainage was established, a rise in intracranial pressure occurred and hydration of the central nervous system was found. There was diffuse swelling of the brain and the fluid was seen to be more prominent in certain places, such as the ventricles and central canal, the chorioid plexus, the perineuronal spaces and the perivascular channels. When drainage was established and free escape of cerebro-spinal fluid was allowed, there was little rise in intracranial pressures and no recognizable hydration of the central nervous system occurred. No indication is given as to how the intracranial pressure was measured. As far as hydration is concerned, it is stated that the ventricles and central canal remained undistended, the chorioid plexus was free of vacuoles and the perineuronal spaces and perivascular channels were not dilated. Kubie concludes that lowering of osmotic pressure of the blood by the intravenous administration of hypotonic solutions causes a transudation of fluid from the capillaries into perivascular and perineuronal areas throughout the whole of the central nervous system and that by simultaneous drainage of the cerebro-spinal fluid a free escape of this transudate is accomplished from the depths of the tissue to the surface through some pathway of least resistance, presumably the perivascular system. He states that the washing out of perivascular exudates into the subarachnoid space has been achieved in experimental meningo-encephalitis in cats (this work was done by Kubie and Shults) and he suggests that forced drainage may be a means of treatment of certain infections of the central nervous system in man.

This work by Kubie appeals to the imagination and merits close study. When hypotonic saline solution is introduced into the circulation, osmosis will take place into all the tissues of the body. Kubie did not measure the cerebro-spinal fluid which he removed from the subarachnoid spaces of his animals. This would have given some information as to the amount which was retained in the tissues other than those of the central nervous system. The animals were all killed and it was thus impossible to measure the urine and to weigh the animals so that an estimate might be made of the time which would elapse before the animal returned to its previous normal state. The animals experimented upon were normal animals, but the photomicrographs reproduced from the work of Kubie and Shults shows that the lymphocytes were extruded from the perivascular spaces of a cat suffering from experimental meningo-encephalitis and were lying in the subarachnoid space. Kubie's report (previously mentioned) which is shortly to be published on observations made on patients, will be awaited with interest.

There is another point to which reference should be made. In the chorioid plexus of animals which had not been subjected to drainage, huge water-filled vacuoles were found. Kubie states that the significance of this is not clear. He suggests two possibilities. Either it is not merely the passage of water through the plexus which produces the vacuolation, but rather a joint effect of this and of the increasing intraventricular pressure or else when the cerebro-spinal fluid is allowed to drain during the injection, the passage of water from the blood stream into the cerebro-spinal spaces takes place, not through the plexus alone, but more generally through all the capillaries of the central nervous system. From the conclusions previously mentioned it may be stated that Kubie is rather inclined to accept the second view. Dandy has shown that the chorioid plexuses are the sole source of the cerebro-spinal fluid, at any rate under normal conditions. The work of Hailiburton, Mestrazat and Kinura has indicated that the fluid is a dialysate and that the cells covering the chorioid plexus exercise some special selective action. Kubie did not analyse the fluid which he withdrew from his animals; this might have yielded some useful information. Cushing has expressed the opinion that there may be a double source of fluid, from the chorioid plexuses and from the perivascular spaces which surround the blood vessels dipping into the brain from the *pia arachnoid*. If it be granted that both methods of secretion or excretion of cerebro-spinal fluid occur in these peculiar circumstances, it is difficult to understand how much osmosis from the circulation into the cerebral tissues could take place without a corresponding rise in intracerebral pressure. Kubie has stated that there is no significant rise when drainage is established, but it is a little difficult to see how he has determined this. The estimation of the venous pressure alone will not give him the information and while cerebro-spinal fluid is escaping, it will be impossible to measure the pressure in the usual way with a manometer. If the views of Smith on poliomyelitis are correct, the increase in intracranial pressure in that condition would be disastrous. Although it cannot be granted that Kubie has definitely established his thesis, the findings of Kubie and Shults in cats show that some useful results may be expected and, as previously mentioned, Kubie's unpublished results may throw further light on the subject. In the meantime caution must be observed before this method of treatment is applied to human beings. On paper at any rate the procedure is not free from danger and as far as results are concerned, it is easy to mistake a *post hoc* for a *propter hoc*.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Radiotherapy in Gynæcology.

P. Werner (*Wiener Medizinische Wochenschrift*, June 30, 1928) gives his experiences with radiotherapy in various gynæcological lesions. Patients with cervical carcinoma generally manifest considerable improvement after such treatment. Unfortunately in the majority of instances these good results do not last more than one to three years when cachexia, recurrence of the tumour and frequently urinary fistulæ develope. Therefore all patients with operable tumours should be operated upon and radiotherapy reserved for patients with inoperable tumours and those presenting some contraindication to operation. X rays are to be preferred to radium for post-operative treatment in all cases. The treatment of recurrences after the radical operation is on the whole very disappointing. The author has had good results from the use of radium in carcinoma of the body of the uterus. There are dangers, however, from uterine perforation or possible spread of infection following on the intrauterine manipulations. Carcinoma of the vagina and vulva give equally bad results following either surgical treatment or radiotherapy. In all cases of ovarian carcinoma the author drains away the ascitic fluid and removes as much of the tumour and secondary growths as possible. X ray therapy is then given and the results have been very encouraging. In non-malignant uterine hæmorrhage, owing to the difficulty of estimating the effects on the ovaries, radiotherapy is no longer employed for young women, but only for those at the menopause. Provided carcinoma has been excluded by a diagnostic curettage, radiation of the ovaries is performed for all hæmorrhage at the menopause. The patient should be warned that the following one or two periods may be excessive, but that amenorrhœa will then ensue. Myomata should be thus treated only if the symptoms comprise nothing else than excessive but regular hæmorrhage. The treatment continues until absolute amenorrhœa is obtained. Tumours as large as the closed fist may disappear altogether, while larger ones are considerably diminished in size. Radiation of the pituitary gland is reserved for young women with excessive menstrual hæmorrhage. It is also of value for patients with obstinate dysmenorrhœa. In some instances radiation of the thyreoid gland in addition is helpful. Radiation of the spleen will increase the coagulability of the blood. This effect is rapid in onset, but does not last for long. Therefore it is useful in combination with radiation of the pituitary gland which takes some time to act. The author has used it also in combination with radiation of the ovaries to control the hæmorrhage which may occur before amenorrhœa is established. This treatment is also valuable in the pre-operative treatment of hæmophilic patients and also for *melæna neonatorum*. Radiation of the liver should be combined with it in difficult cases. Tuberculosis of the genital tract and of the peritoneum reacts very favourably to radiation. While amenorrhœa necessarily follows the dosage required in such conditions, it must be remembered that the anatomical changes, generally present, preclude any possible future pregnancy. The prognosis in these circumstances must be based on the general condition of the patient and not alone on the pelvic examination. Carefully graduated doses have a definite effect on inflammatory conditions of the uterine appendages. This acts partly by reducing the monthly loss of blood and partly by a direct effect on the local lesion. Similarly early mastitis may be prevented from reaching the suppurative stage. *Pruritus vulvæ* should be treated with the hard filtered rays of deep therapy and not the soft ray used by the dermatologist. The course should consist of ten daily doses of small capacity. Unfortunately *kraurosis vulvæ* does not react to the same extent as pruritus and the treatment has to be repeated at frequent intervals. However, the patient is much more comfortable than after a severe plastic operation.

Pathology and Treatment of Sterility.

F. Weinfeld (M.D. Thesis, University of Giessen, 1927) has analysed 287 cases of sterility from the point of view of causation, treatment and results. In 20% there was a definite hypoplasia of the genital organs. Ninety patients manifested general constitutional disturbances, such as infantilism, asthenia and adiposity. Endocrine defects, mainly of the thyreoid gland, were noted in 79 instances. Combined with the former group, this represents 58% of all the cases and emphasizes the important ætiological bearing of general diseases on sterility. Inflammatory lesions, especially appendicitis, were noted in 25% and tuberculosis of the genital tract and peritoneum in 4·7%. Adhesions and inflammatory lesions of the tubes and ovaries were seen in 44 persons and in many of these they were probably due to a primary appendicitis. Uterine displacements played a very small part and in only 20 instances was a retroflexion considered to be responsible for the sterility. Degenerative changes, mainly cystic, of the ovaries were noted in 58 persons and although in some the condition may have been primary, the majority was secondary to endocrine or general disturbances. Thirty-five patients with genital hypoplasia or constitutional disease were cured. Success was obtained with nine of fifteen patients with uterine displacement. The presence of tubal inflammation was an important factor in this group. Forty-five patients were treated by insufflation of the tubes and 25% were cured.

Diagnosis of Extrauterine Pregnancy.

B. A. Iwanoff (*Monatsschrift für Geburtshilfe und Gynäkologie*, May, 1928) discusses the value of several tests in the diagnosis of extrauterine pregnancy. He considers that the presence of hæmatin in the blood serum is not of great importance in the diagnosis of an unruptured extrauterine pregnancy, although in many instances a reaction is obtained. A response to the blood sedimentation test requiring one hour or longer, a slight leucocytosis and a drop in temperature are generally indications that the ectopic pregnancy has not ruptured and help in the differential diagnosis from salpingitis. Puncture of the posterior fornix and the presence of blood do not always mean that an ectopic pregnancy has ruptured. This may be due to ovarian hæmorrhage, a volvulus or hæmorrhagic transudation from an ovarian cyst. If it be associated with leucocytosis and a prolonged response to the sedimentation test be noted, considerable value is attached to it. The author emphasizes the value of a detailed history and the elicitation of all the physical signs which still are of more importance in the diagnosis than any biochemical test.

End Results of Ovarian Carcinoma.

E. Schleyer (*Monatsschrift für Geburtshilfe und Gynäkologie*, July, 1928) summarizes the end results of patients with ovarian carcinoma treated during 1910 to 1925 in the First Clinic at Vienna. There were 115 patients in all, including 18 for whom an exploratory laparotomy alone was performed. The primary operative mortality among the 97 patients was 12·37%. Of those who recovered, 21% had a relative freedom from recurrences and only 9·5% an absolute cure after five years' observation. The best results were obtained with patients suffering from carcinomatous degeneration of pseudo-mucinous ovarian cysts and the worst with those suffering from metastatic ovarian growths. Serous papillary cysts and the solid type of carcinoma occupied an intermediate position. In all cases after the abdomen was opened and the growth inspected, a decision was made regarding the operative procedure. In the majority of patients total removal of uterus and appendages was performed. In unilateral malignant disease of an ovarian cyst, especially if the tumour could be removed without spilling the contents, the opposite ovary was left, provided it appeared to be healthy. The prognosis cannot be expected to be good because of the difficulty of early diagnosis. Most patients did not present themselves until palpable tumours and often metastases were present. The author recommends that all tumours of the ovary should be removed and examined in order to prevent possible malignant degeneration later. Even if the con-

dition be inoperable, the removal of much of the mass renders post-operative radiotherapy more likely to be successful.

OBSTETRICS.

Treatment of Puerperal Sepsis.

G. ASCHERMANN AND L. ROSENBLUM (*Monatsschrift für Geburtshilfe und Gynäkologie*, July, 1928) give their experience with the method of local immunization described by Besredka in the treatment of puerperal sepsis. A uterine culture is obtained and an autogenous vaccine is prepared from the organisms present. The uterus is douched with a non-irritating solution, such as normal saline solution and strips of gauze are wrung dry out of a mixture of the vaccines obtained from the patient and are packed into the uterine cavity and vagina. The authors emphasize the importance of thoroughly packing the uterus and not merely inserting a strip of gauze through the cervix or placing a tampon in the vagina against the cervix. The packing is repeated every day until definite improvement results. The uterine douche can be omitted after the first day. Definite relief from pain is noted at once and there is a fall in temperature within a short period. Details of six cases are given with one death. They were all severe infections and did not come under control before the end of the first week of the puerperium. Naturally, treatment should be commenced at an earlier stage, if possible, in order to obtain the maximum amount of success.

Interstitial Pregnancy.

Z. SZATHMÁRY (*Monatsschrift für Geburtshilfe und Gynäkologie*, July, 1928) describes a case of interstitial pregnancy at term. He states that most interstitial pregnancies encroach during development either on the lumen of the Fallopian tube or towards the uterine cavity. In the rarest form, as illustrated by the author's case, the pregnancy is confined to the interstitial portion of the tube. The patient was a *primipara* and was admitted with a history of weak labour pains for six hours. The fœtus lay in an apparently normal position. Several hours afterwards there was a sudden attack of abdominal pain, collapse and profound anæmia. There was considerable tenderness on palpation and a disappearance of the fœtal heart sounds. The cervix admitted one finger, the membranes were intact but no presenting part could be felt. A provisional diagnosis of uterine rupture due to some developmental anomaly of the uterus was made. On opening the abdomen a foot was found presenting and a full term still-born child was extracted. The clinical picture at this stage resembled that of a bicornuate uterus. Supravaginal hysterectomy was performed. It was concluded from the position of the pregnancy in relation to the tube and round ligament as well as from the histological finding of no true decidual cells in the region of the placenta that it was a true interstitial pregnancy.

Rupture of Cæsarean Section Scar.

H. KÜSTNER (*Monatsschrift für Geburtshilfe und Gynäkologie*, July, 1928) discusses the causation of rupture of the uterus following a previous Cæsarean section or other operation or injury to the uterine wall. This risk may also be incurred in pregnancies following perforation of the pregnant or non-pregnant uterus during curettage or dilatation. Owing to the number of patients with *placenta prævia* and eclampsia who are delivered by operation, the risk of rupture of the scar has greatly increased within recent years. An important factor in the prognosis is the site of rupture. It is more serious when occurring in the upper segment than in the cervix or the lower uterine segment. Scars in the body of the uterus are in close association with strongly contracting muscular fibres and if any obstacle to delivery exists, there is a greater risk of rupture. The rupture is not confined to the zone of the scar, but extends freely into the thick muscle surrounding it. This risk of rupture is a strong indication for the use of the lower segment operation in place of the classical section. Of even greater importance is the interval which has elapsed between the operation and the next pregnancy. He considers that at least one year should elapse before a further pregnancy is considered.

The Duration of Pregnancy.

H. TERRAHE (M.D. Thesis, University of Giessen, 1927) has analysed 3,000 birth records and has paid particular attention to 247 cases in which conception and the date of birth have been accurately calculated. The duration of pregnancy can be estimated either from the end of the last menstrual period or from a definite coitus. The difference between these two methods is on an average 15·4 days. The author takes a standard for a normal child one of over 2,800 grammes in weight, 48 centimetres in length and with a mento-occipital circumference of 36 centimetres. Investigation of these 247 pregnancies shows that the average duration was 270 days. The minimum was 250 days and the maximum 293 days. He considers that it is impossible to fix any definite standard for legal purposes. Statistics show that normally developed children may be born from 210 to 245 days after conception as well as those corresponding to 300 or more days. The weight and length of the child do not assist, for various authors have shown that a big child may follow a short period and similarly prolonged pregnancy may not be associated with a big child. From his summary he concludes that although most children are born 270 days after conception and a few between 240 and 250 days, yet a sufficient number of even earlier births has been noted to make the legal aspect one in which each case must be decided upon its own merits.

The Children of Eclamptic Mothers.

D. S. PLOTKIN (M.D. Thesis, University of Giessen, 1927) reviews the histories of the children of eclamptic mothers. From 1900 to 1925 there were 123 patients with eclampsia with 138 children born at the obstetric clinic of the University of Giessen. Thirty-six children were still-born and fourteen died within the first ten days —a total mortality of 36·2%. There were thirty-one premature births with twelve still-born and six neo-natal deaths—a total mortality of the premature infants of 58·5%. As was to be expected, the results of the conservative method of treating eclampsia as regards the infant were not so good as those following active measures of delivery. The author's figures show that, provided the first dangerous ten days of neo-natal life are passed, the chances of survival are not influenced by the previous eclamptic condition of the mother. The future life history of 87 children was obtained by means of a *questionnaire*. From this it was shown that no nervous nor cerebral symptoms had occurred. Although many of the children were handicapped at the onset of life by prematurity, asphyxia and artificial feeding, these did not prevent normal development up to school age. Finally he considers from his own statistics as well as from those of others, that breast feeding should always be insisted upon.

Effect of X rays on the Fœtus.

K. KAISER (*Monatsschrift für Geburtshilfe und Gynäkologie*, May, 1928) describes the condition of an infant whose mother had been treated with the deep X ray therapy when five months pregnant. There was a history of removal of one ovary for carcinoma three years previously. When first seen, she was five months pregnant and there was a mass of recurrent malignant disease in the pelvis and abdomen. Abortion could not be performed because of the difficulty of access to the cervix. Therefore, a course of deep X ray therapy was given. She had a normal delivery at term, although the infant was somewhat under normal weight. The child had a head slightly smaller than it should be. In addition there were nævi on the forearm and thighs as well as a definite pigmentation of the skin. The weight of the child may be explained by the poor general condition of the mother. The author is not sure whether the nævi have any connexion with the radiotherapy. The infant is taking nourishment well and apparently growing in a normal fashion. Further observation will be necessary to determine whether any other defects will develope.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Women's Hospital, Carlton, on July 18, 1928. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Pathological Specimens.

DR. C. H. MOLLISON showed a series of pathological specimens illustrating rare and interesting pathological conditions found at operation and *post mortem* examination.

Bony Deformity of the Pelvis.

DR. IVON HAYES showed two patients with pelvic bony deformity. The first patient was a *primipara*, aged twenty-two years, a typical achrondroplasiac. Labour had commenced at 10 p.m. on July 13, 1928, the membranes rupturing on the patient's admittance at 7 p.m. the following evening. The external measurements of the pelvis were: interspinous 23 centimetres, intercristal 24·5 centimetres and external conjugate 16 centimetres. On vaginal examination the obstetrical conjugate was found to be approximately four centimetres. Cæsarean section had been done at 9.20 p.m. and a living male weighing 3·2 kilograms was delivered. The size of the conjugate was an absolute indication for section, even had the child been dead, as vaginal delivery was impossible.

Dr. Hayes's second patient was a woman, aged twenty-four years, who had borne two children. This patient was of very short stature and presented a definite deformity of the spine, an acute lordosis. In March, 1926, a Cæsarean section had been performed, apparently without a trial of labour, and a child of 3·4 kilograms was born. Labour began at 11 p.m. on July 7, 1928, the pains not being very strong. The external pelvic measurements were: interspinous 22·5 centimetres, intercristal 23 centimetres and external conjugate 17·5 centimetres. As there did not seem to be any disproportion between the head and the pelvis, labour had been allowed to go on. At 6 p.m. on July 10, 1928, the membranes had ruptured and two hours later the child which weighed 2·7 kilograms (six pounds) was born without any difficulty.

Dr. Hayes pointed out that these two cases illustrated the uncertainty of diagnosis from external measurements and in the second patient the value of a "trial of labour" when pronounced disproportion was not evident.

Vaginal Hysterectomy.

DR. W. CUSCADEN showed several patients on whom the operation of vaginal hysterectomy had been performed. Dr. Cuscaden pointed out that the site of operation had returned to a condition so nearly normal that it was difficult to see that any operation had been performed. He particularly drew attention to the solidity of the pelvic floor and the small effect that coughing and straining had upon it.

In many English schools vaginal hysterectomy was looked upon with disfavour, whereas its popularity caused it to be done for unsuitable conditions in some Continental clinics.

Dr. Cuscaden's technique was as follows. The parts were freely painted with 5% iodine, the cervix was drawn down with American bullet forceps and plugged with a piece of gauze. The cervix was outlined with a diamond-shaped incision and the dissection carried up between the bladder and the uterus. Most of the dissection was done with a sharp scalpel, the pubo-cervical ligament was split and it had to be remembered that some of the superficial bladder musculature was continuous with the cervix. The pointed bottom of the utero-vesical pouch was exposed and the peritoneum opened. The fundus was delivered into the vagina and drawn strongly to one side. The broad ligament of the opposite side was then tied with catgut and cut down as far as the internal *os*. By dissection with the end of a blunt pair of scissors between the layers of the broad ligament the uterine artery was traced to the side of the uterus. The uterine artery was tied and cut. The proximal stump of the artery with the peritoneum and with some cellular tissue was stripped laterally, the ureter being carried with it. After performance of a posterior colpotomy a long clamp was applied to the transverse ligament of the cervix. The same procedure was adopted on the opposite side. The transverse ligaments were then cut and the uterus removed. The two long clamps were laced to each other with chromicized catgut, the clamps were slipped out and the lace pulled tight; thus the two transverse ligaments were brought together. The two broad ligaments were either sewn to each other or oversewn. The pubo-cervical ligaments were found in the anterior angle of the incision and sewn to the transverse ligaments. The vaginal mucosa was carefully sutured, as it was inclined to bleed. No special after-treatment was necessary.

Radiography and Fœtal Development.

Dr. Cuscaden also showed a skiagram from a woman of thirty-five years of age, who had had three abortions with an intrauterine death of the fœtuses. An examination had been made ten and a half months from her last menstrual period. Seven and a half months before she had had a severe hæmorrhage which was believed to have been a threatened abortion. The patient was short, fat and difficult to examine. The opinion had been expressed that she was seven and a half months pregnant. In the face of her history it had been most important to know if the pregnancy had extended beyond term. The skiagram revealed an epiphysis in the lower end of the femur and a less distinct one in the upper end of the tibia. Labour had been induced and a full-time living child was born.

Colloid Carcinoma of the Sigmoid.

DR. ALLEN ROBERTSON showed a woman of twenty-eight who had been admitted with carcinoma of the sigmoid colon. Enlarged glands had been present all over the mesentery and mesosigmoid. The growth had been excised and an end-to-end anastomosis performed. A microscopical examination of the growth revealed a colloid carcinoma and the glands manifested chronic inflammatory changes only.

Vesico-Vaginal Fistula.

Dr. Robertson's second patient was a young woman of twenty-two who had sustained an extensive laceration of the anterior vaginal wall during childbirth with the formation of a large vesico-vaginal fistula.

Two attempts at vaginoplasty had improved the condition and narrowed the fistula. There was no dribbling of urine while the patient was lying down. This occurred when the patient assumed the erect position.

Biliary and Renal Calculi.

Dr. Robertson also showed X ray photographs of biliary and renal calculi.

Results of Salpingostomy.

DR. B. MILNE SUTHERLAND showed a patient who was suffering from probable missed abortion and on whom the operation of salpingostomy had been performed two years before.

Other Demonstrations.

Among others who showed patients were Dr. J. S. Green, Dr. W. D. Salteau, Dr. A. Sherwin and Dr. P. S. Brett.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Rau, Noel, M.B., B.S., 1927 (Univ. Sydney), 67, Birriga Road, Bellevue Hill.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Buchanan, Andrew Russell, M.B., B.S., 1927 (Univ. Melbourne), Alfred Hospital, Prahran.
Gault, Edward Woodall, M.B., B.S., 1928 (Univ. Melbourne), Auburn.
Woodruff, Harold Addison, M.R.C.S. (England), L.R.C.P. (London), 1912, University of Melbourne.
Brenton, Vernon Brentani, M.B., B.S., 1926 (Univ. Melbourne), Port Melbourne.
Brittingham, Lindsay Charles, M.B., B.S., 1928 (Univ. Melbourne), Hawthorn.
Thomas, David Lewis Gordon, M.B., B.S., 1928 (Univ. Melbourne), Camberwell.

Obituary.

FRANCIS LAWRENCE BIGNELL.

By the death of Dr. Francis Lawrence Bignell which was recorded in a previous issue of this journal, the medical profession has lost one who put the safety of others before his own welfare and who was always actuated by a sense of duty.

Francis Lawrence Bignell was the son of Margaret A. Bignell and of the late Edmund Bignell. He was born at Carlton, Victoria, on May 3, 1886. His education was begun at the University High School and later on he became a pupil at South Melbourne College. He studied medicine at the University of Melbourne and became a resident student at Queen's College. He was fond of sport. At school he was awarded a silver and then a gold medal for sports and he played football with his school team. At the University he played lacrosse and represented the University in several matches; for this he gained his blue.

After graduation as Bachelor of Medicine and Bachelor of Surgery Bignell went to Murray Bridge in South Australia, where he worked with Dr. McQuarrie until he was called to the Brisbane General Hospital to fill a resident post. In 1912 he took up practice in Lismore, New South Wales. He was interested in military affairs and became Captain and then Major in the Army Medical Corps of the Commonwealth Military Forces. On the outbreak of war it was some time before he was allowed to leave Australia. Finally in 1916 he sailed as Regimental Medical Officer to the Twenty-sixth Battalion. At the front his devotion to duty attracted notice. He was twice mentioned in dispatches, was promoted to the rank of Major and received the Distinguished Service Order. This distinction was conferred on him for his work at Millencourt. Dr. H. L. St. Vincent Welch in the tribute which is published below, bears testimony to his heroism. He was severely gassed and although he recovered sufficiently to be able to resume work at a casualty clearing station, his health was permanently affected and his untimely death was hastened. He returned to Australia in 1919 and took up again his practice at Lismore where he remained till the time of his death. The sympathy of the medical profession and particularly of former members of the Australian Imperial Forces, is extended to his widow.

Dr. H. L. St. Vincent Welch writes:

It is with great regret that I have heard of the death of F. L. Bignell. His untimely end was undoubtedly the delayed result of an action which I feel should be more widely known than it may be at present.

In May, 1918, Major Bignell, then one of my officers in the Sixth Australian Field Ambulance, was in charge of a dressing station in the village of Millencourt. During the night following the Battle of Treue the enemy bombarded Millencourt with, according to the Divisional Headquarters estimate over twelve thousand shells of all calibres, high explosive, gas and universal.

The gas-proofed cellar of the dressing station was blown in and set on fire and Major Bignell continued throughout the night to work in the open.

Finding it impossible, on account of the darkness, smoke and dust, satisfactorily to carry on his work while wearing his respirator, Bignell deliberately took it off, knowing full well what the result must be.

By morning he was gassed, blinded and terribly burned by mustard gas and for some time it appeared doubtful whether he could live.

Major Bignell finds a place among those very gallant gentlemen who did their duty, not counting the cost.

PETER HANNAH CUNNINGHAM.

It is with regret that we have to record the death of Dr. Peter Hannah Cunningham after a short illness on August 7, 1928.

Peter Hannah Cunningham was born in Glasgow and received his early education at the Presbyterian Normal School. After leaving school he entered the Glasgow University and studied medicine. He took the degrees of Bachelor of Medicine and Master of Surgery in the year 1879. He was contemporary with John Glaister, Eric Sinclair and James Dunlop Boyd. As a student he came under the influence of the late William Tennant Gairdner, for whom he had the most profound reverence and veneration. Spurred by his precept and stimulated by his teaching, Cunningham achieved signal success as a student and gained the confidence of his teachers and comrades. After graduation he gained a house appointment at the Western Infirmary and here again he was associated with Tennant Gairdner. The influence of his famous teacher was discernible throughout the whole of his professional life. After his term of office at the Western Infirmary he was appointed a house physician at the Bournemouth Hospital. In 1880 he left England for Australia and on his arrival in Victoria he selected the town of Talbot. He worked assiduously and without stint for the residents and for forty-three years he was their staunch friend and adviser, their trusted doctor and in many matters their chosen leader. From the early days he took an active part in the affairs of the Amhurst District Hospital. In the early 'eighties the teachings of Lister were making their way to Victoria and Cunningham had a strenuous and uphill fight to secure recognition for the antiseptic method at the hospital. In addition to his hospital and private practice he concerned himself with hygiene and became the health officer for the Borough and Shire of Talbot and for the Shire of Lexton. In those days public health work had not emancipated itself from the realm of water supply, disposal of excrement and removal of house wastes. Preventive medicine was a dream. Notwithstanding the limited scope of the work, Peter Hannah Cunningham performed his duties with characteristic thoroughness and energy. In 1903 he and the late A. Gresswell strove against many obstacles to establish in connexion with the Amhurst District Hospital, a sanatorium for the treatment of tuberculous patients. At first the old fashioned military bell tents were used for the accommodation of the patients. These were soon found to be unsuitable and were replaced by four bed tents. Still later Cunningham persuaded the board to construct bungalows. At the time of his retirement in 1923 he was advocating improvements in the water supply, the sanitation and lighting of the institution. For over twenty years he was also an honorary surgeon at the Maryborough Hospital.

Peter Hannah Cunningham was a very unassuming man with simple tastes and a great dislike for publicity. He was widely read and had an almost inexhaustible store of general knowledge. He was an enthusiastic student of the classics and was as much at home with Plato as he was with Emerson, Matthew Arnold, Robert Louis Stevenson or Charles Darwin. Gardening and ethnology were his chief hobbies. For the greater part of his life he was an early riser and he loved to spend time and trouble in his garden before breakfast, among his roses and daffodils. His garden claimed much of his attention; he loved it and as a result it was widely known and admired. He knew all the "blackfellows' ovens" in the district and in the course of time he collected a large variety of stone implements, including knives, hammers, axes and grinding stones. He was a lover of birds and had a considerable knowledge of ornithology. Samuel Gee once said: "A man's worth is to be estimated by the value

of the objects which he desires and seeks after. As he loves, so he lives." It was thus with Peter Hannah Cunningham. He leaves a widow, one son and two daughters.

DONALD CLARK.

WE regret to announce the death of Dr. Donald Clark which occurred at Sydney on October 12, 1928.

EGBERT FLORANCE.

WE announce with regret the death of Dr. Egbert Florance which occurred at Cootamundra, New South Wales, on October 13, 1928.

Books Received.

PHARMACOPŒIA AND REFERENCE HANDBOOK OF THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY; Fourth Edition; 1928. Sydney: The Australasian Medical Publishing Company, Limited. Demy 32mo., pp. 98.

A HANDBOOK ON VENEREAL DISEASES, by W. Turner Warwick, F.R.C.S.; 1928. London: Faber and Gwyer, Limited. Crown 8vo., pp. 232, with illustrations.

Diary for the Month.

Nov. 1.—South Australian Branch, B.M.A.: Council.
Nov. 2.—Queensland Branch, B.M.A.: Branch.
Nov. 6.—Tasmanian Branch, B.M.A.: Council.
Nov. 7.—Western Australian Branch, B.M.A.: Council.
Nov. 8.—Victorian Branch, B.M.A.: Council.
Nov. 9.—Queensland Branch, B.M.A.: Council.
Nov. 13.—Tasmanian Branch, B.M.A.: Branch.
Nov. 13.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 14.—Victorian Branch, B.M.A.: Branch.
Nov. 15.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 16.—Eastern Suburbs Medical Association, New South Wales.
Nov. 19.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 20.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments.

Dr. Hugh McIntyre Birch (B.M.A.) has been appointed Acting Deputy Superintendent, Parkside Mental Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Resident Registrar, Three Assistant Resident Medical Officers, Honorary Clinical Assistant.

INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Consultant Surgeon.

MARRICKVILLE DISTRICT HOSPITAL, NEW SOUTH WALES: Junior Honorary Medical Officer and Anæsthetist.

MARYBOROUGH HOSPITALS BOARD, MARYBOROUGH, QUEENSLAND: Junior Resident Medical Officer.

NATIONAL ASSOCIATION FOR THE PREVENTION AND CURE OF CONSUMPTION: Honorary Medical Officer.

REPATRIATION COMMISSION, VICTORIA: Junior Resident Medical Officer.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN, NEW SOUTH WALES: Honorary Assistant Surgeon.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Physician.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. Marrickville United Friendly Societies' Dispensary. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor." THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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